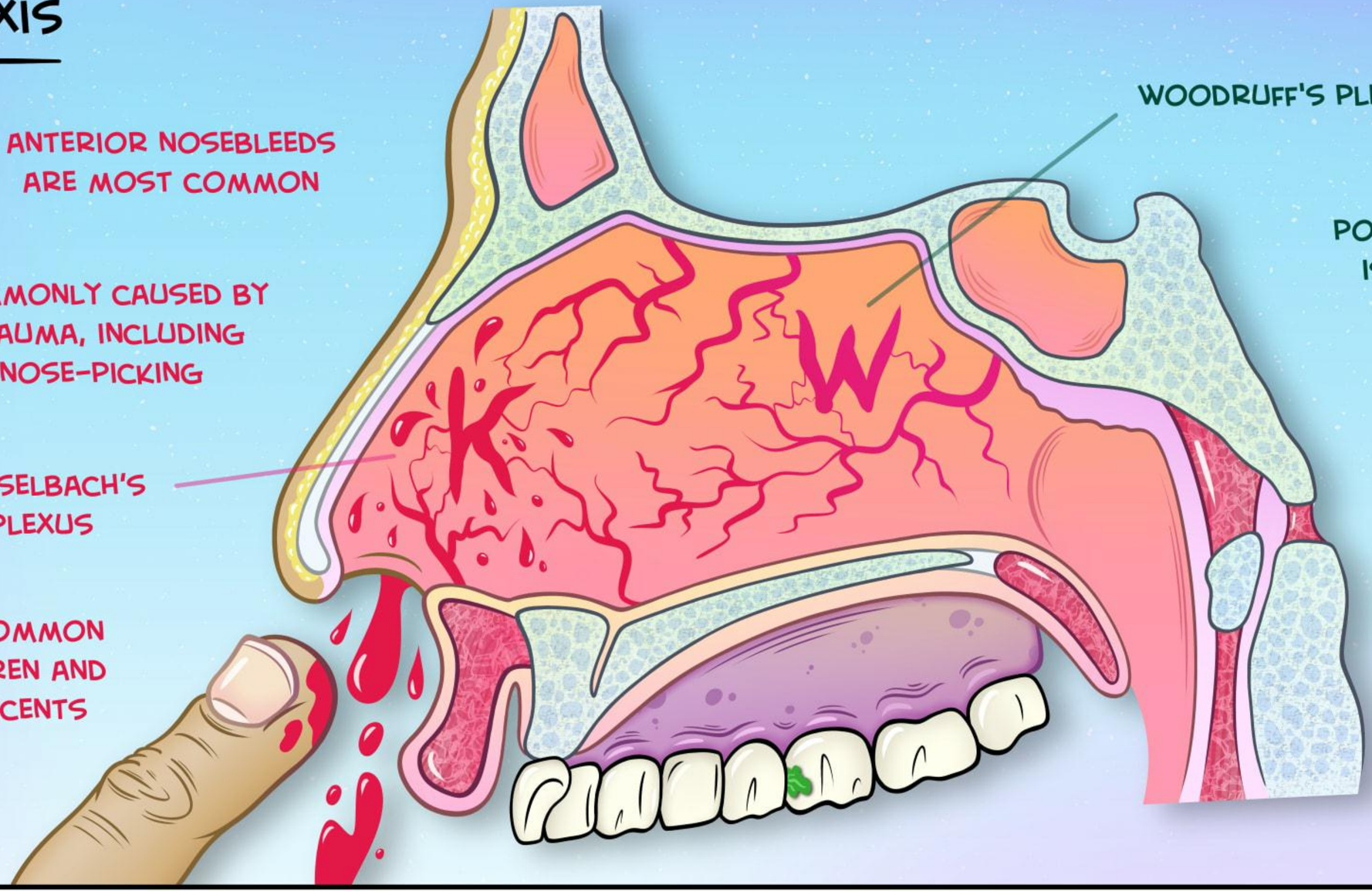
EPISTAXIS

ANTERIOR NOSEBLEEDS ARE MOST COMMON

COMMONLY CAUSED BY TRAUMA, INCLUDING NOSE-PICKING

KIESSELBACH'S PLEXUS

MORE COMMON IN CHILDREN AND ADOLESCENTS



WOODRUFF'S PLEXUS

POSTERIOR BLEEDING IS LESS COMMON

ASSOCIATED WITH HYPERTENSION AND ATHEROSCLEROSIS

USUALLY SEEN IN THE ELDERLY

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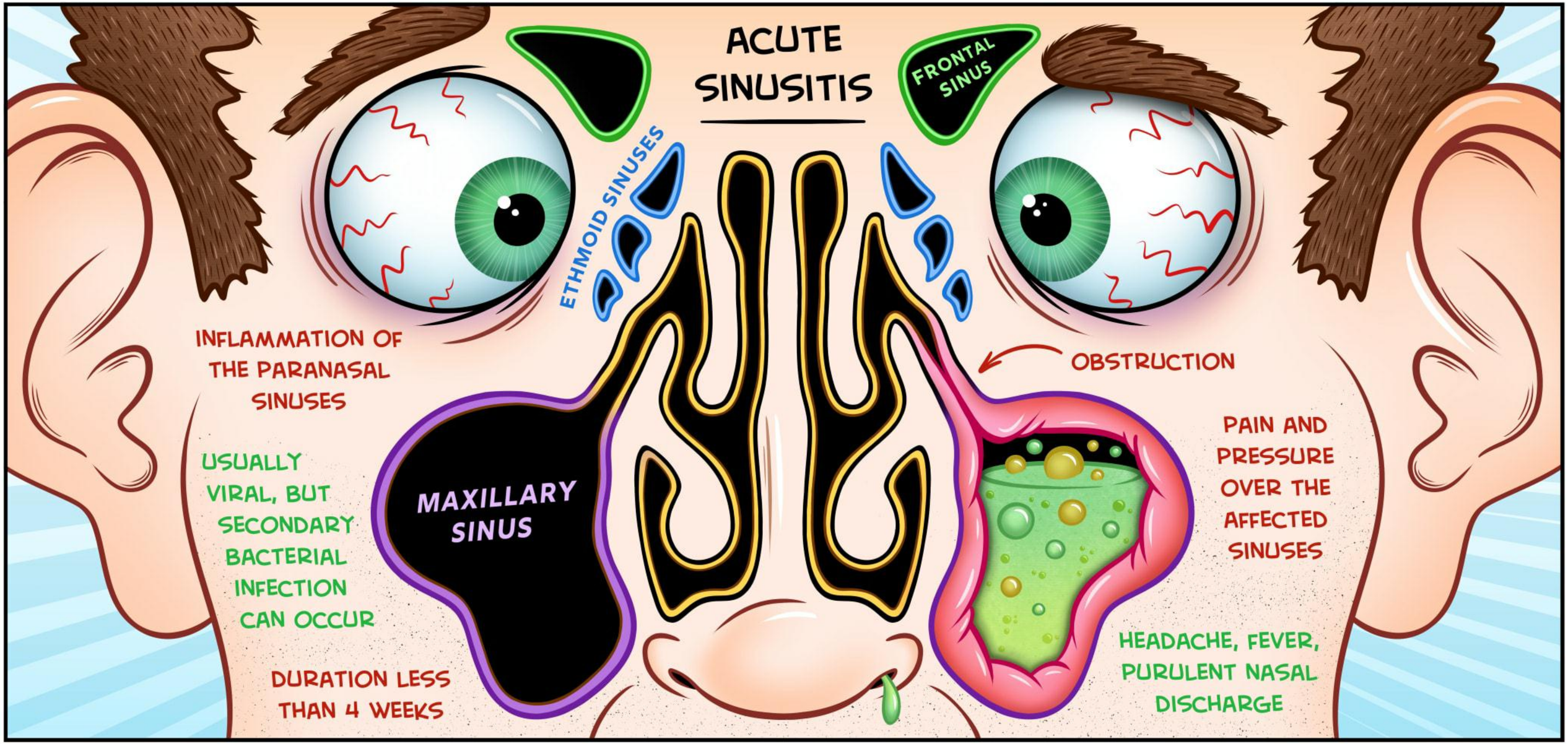
- Epistaxis may be classified as anterior or posterior nosebleeds.
- Anterior nosebleeds are most common, usually occurring at Kiesselbach's plexus in the anterior nasal septum.
    - Kiesselbach's plexus consists of the anterior ethmoid, greater palatine, sphenopalatine, and superior labial arteries.
    - More common in children and adolescents.
    - Commonly caused by trauma, including nose picking.
- Approximately 10% if epistaxis occurs in the posterior nasal cavity, in an area known as Woodruff's plexus.
    - Woodruff's plexus mainly consists of the the posterior nasal, sphenopalatine, and ascending pharyngeal arteries.
    - Usually seen in the elderly.
    - Associated with hypertension and atherosclerosis.
- Initial management of epistaxis involves positioning the patient leaning forward and applying manual pressure to the anterior nose for 15-20 minutes.
- Other treatment options include topical decongestants (oxymetazoline), topical anesthetics (lidocaine), cautery (silver nitrate, electrocautery), packing, and surgery consisting of arterial ligation for intractable bleeding.



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General Features

- Acute sinusitis is an inflammation of the paranasal sinuses usually due to a viral infection, but secondary bacterial infection can occur.
- Often precipitated by an upper respiratory infection.
    - Inflammation causes swelling of the mucosa leading to obstruction and reduced clearance of mucus.
        - Common bacterial pathogens include: *S. pneumoniae*, *H.influenzae*, and *Moroxella cattarrhalis*.
        - Same bacterial etiology as those for otitis media.

Symptoms and Signs

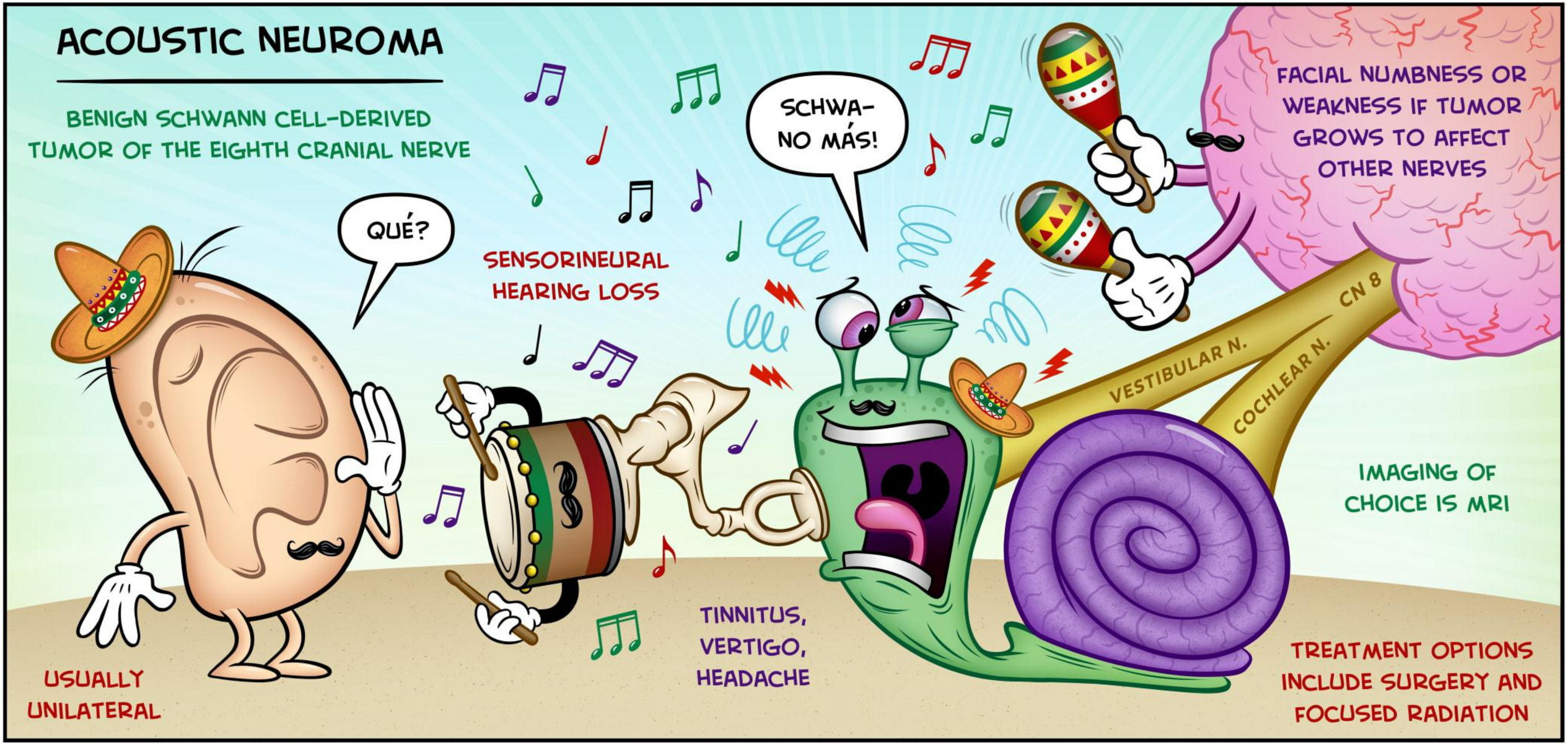
- Pain and pressure over affected sinuses
    - Worse when bending forward
    - Tenderness to palpation
- Headache
- Fever
- Halitosis
- Nasal congestion and obstruction
- Purulent nasal discharge
- Transillumination of the affected sinuses may demonstrate opacification

Diagnosis

- Usually diagnosed clinically.
- Acute bacterial sinusitis should be considered with persistent symptoms lasting longer than 10 days, severe onset of symptoms, or if symptoms worsen.
- CT scan if complications are suspected or in refractory cases.
    - Rules out intracranial or orbital involvement.

Treatment

- Supportive therapy
    - NSAIDs, warm compresses, decongestants, nasal saline irrigation, intranasal corticosteroids
- Antibiotics if symptoms persist for 10-14 days or severe symptoms/signs are present.
    - Amoxicillin-clavulanate



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General Features

- An acoustic neuroma is a benign Schwann cell-derived tumor of the eighth cranial nerve.
- Also known as a vestibular schwannoma.
- Usually unilateral
    - In neurofibromatosis type 2, acoustic neuromas are typically bilateral.

Signs and Symptoms

- Progressive, unilateral sensorineural hearing loss
- Unilateral tinnitus
- Continuous vertigo
- Headache
- Facial numbness or weakness if tumor grows to involve the trigeminal nerve or facial nerve.

Diagnosis and Management

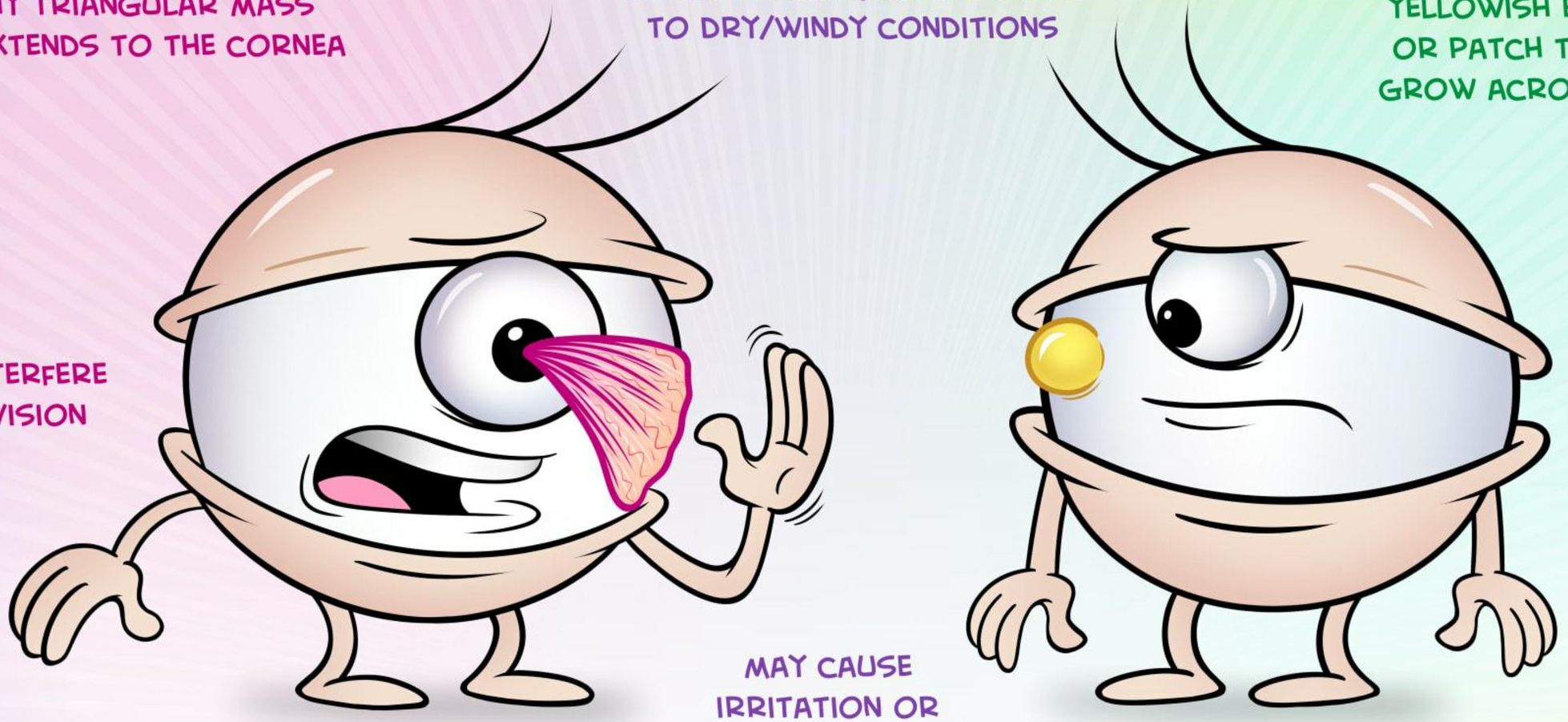
- Imaging of choice is MRI. Vestibular schwannomas can also be detected with CT scan.
- Audiometry may be performed initially to confirm asymmetric sensorineural hearing loss.
- Management may include observation with serial MRIs, surgery, or focused radiation.
    - Treatment depends on many factors including the patient's age and health, severity of hearing loss, and tumor size.

PTERYGIUM

FLESHY TRIANGULAR MASS THAT EXTENDS TO THE CORNEA

MAY INTERFERE WITH VISION

BOTH ARE CONJUNCTIVAL GROWTHS THAT MAY RESULT FROM CHRONIC ACTINIC IRRITATION, REPEAT TRAUMA, OR EXPOSURE TO DRY/WINDY CONDITIONS



MAY CAUSE IRRITATION OR COSMETIC BLEMISH

PINGUECULA

YELLOWISH ELEVATED BUMP OR PATCH THAT DOES NOT GROW ACROSS THE CORNEA

TREATMENT IS NOT USUALLY NECESSARY BUT CAN BE RESECTED

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- Pterygium: fleshy triangular mass that extends to the cornea.
    - May interfere with vision.
- Pinguecula: yellowish elevated bump or patch that does not grow across the cornea.
    - Treatment is not usually necessary but can be resected.
- Both are conjunctival growths that may result from chronic actinic irritation, repeat trauma, or exposure to dry/windy conditions.
- Typically occur on the nasal side.
- They may cause irritation or cosmetic blemish.

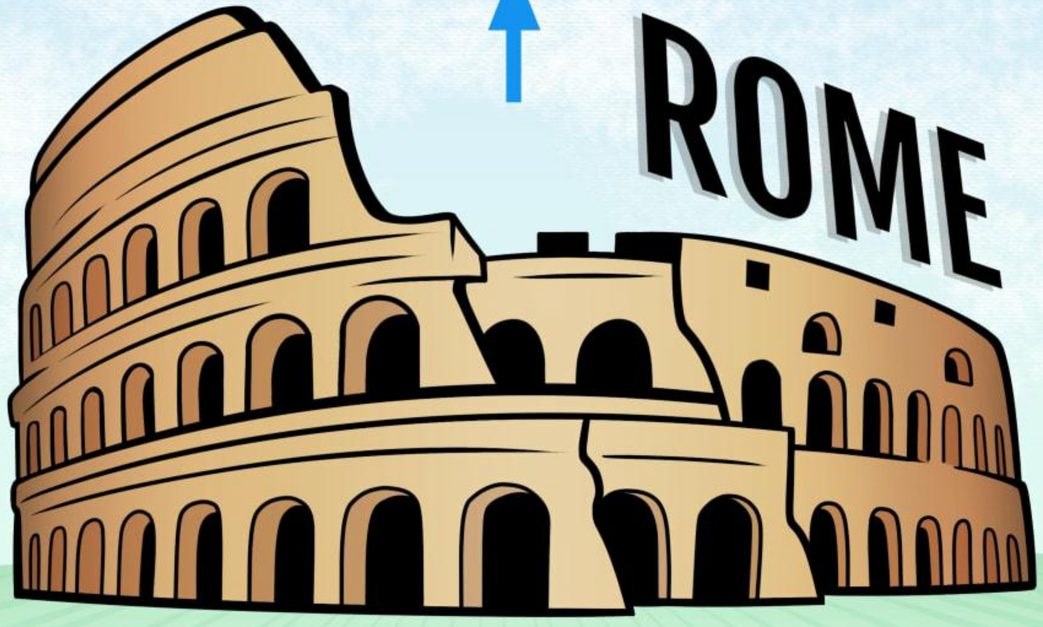
RESPIRATORY

CO_2 ↓ ↑

HIGH PH > 7.45 (ALKALOSIS)

METABOLIC

HCO_3 ↑ ↑



NORMAL PCO_2
35-45 MM HG

NORMAL HCO_3
22-26 MEQ/L

RESPIRATORY OPPOSITE METABOLIC EQUAL

CO_2 ↑ ↓

RESPIRATORY

LOW PH < 7.35 (ACIDOSIS)

METABOLIC

HCO_3 ↓ ↓

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	pH	PCO2	HCO3	Common Causes
Respiratory Alkalosis	↑	↓		Hyperventilation (e.g. in response to hypoxia, metabolic acidosis, increased metabolic demands, pain, or anxiety).
Respiratory Acidosis	↓	↑		Impaired respiration or airflow obstruction which can be caused by a multitude of disorders.
Metabolic Alkalosis	↑		↑	Volume depletion (e.g. vomiting) and diuretic use.
Metabolic Acidosis	↓		↓	High anion gap: ketoacidosis, lactic acidosis, renal failure, toxic ingestions. Normal anion gap: GI or renal HCO3 loss.

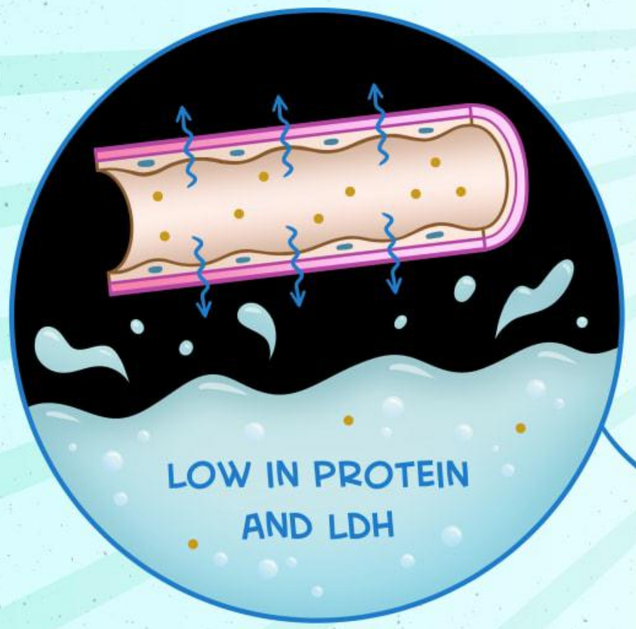
PLEURAL EFFUSION

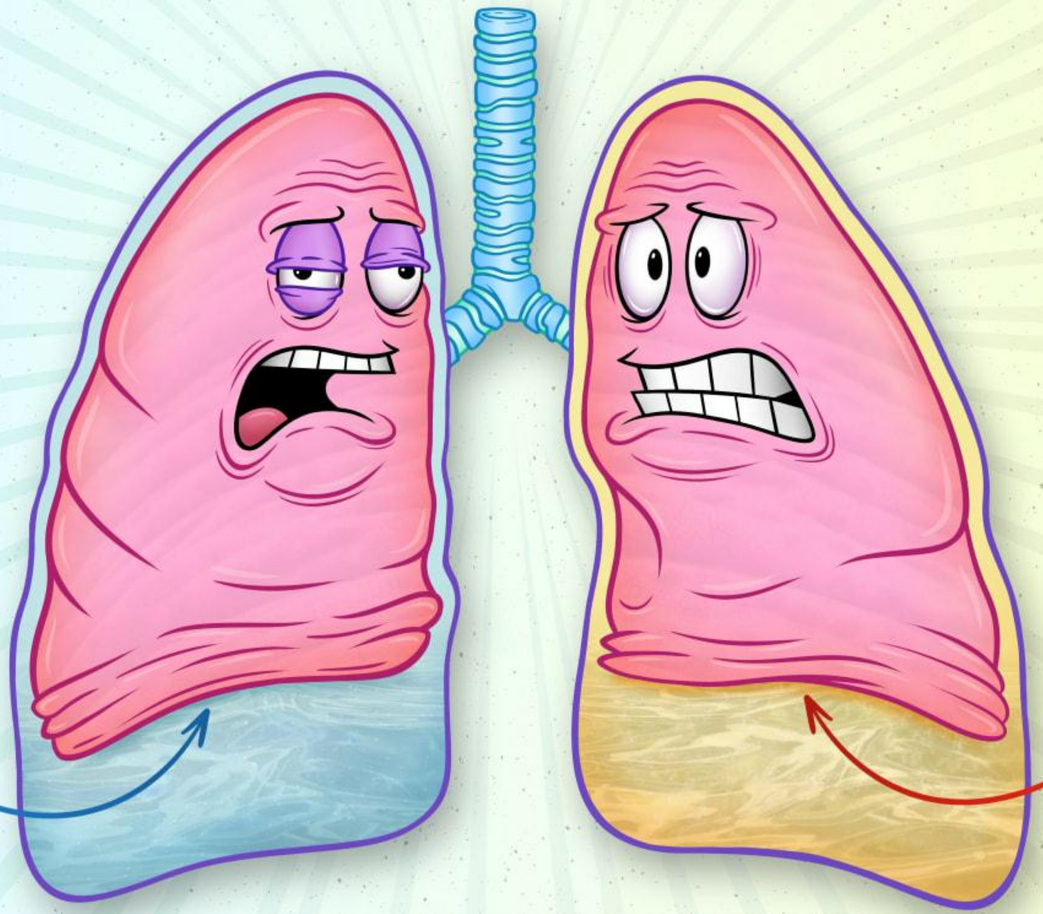
ACCUMULATION OF FLUID WITHIN THE PLEURAL SPACE

TRANSUDATIVE

OCCURS DUE TO INCREASED HYDROSTATIC PRESSURE OR LOW PLASMA ONCOTIC PRESSURE

E.G., CHF, CIRRHOSIS, NEPHROTIC SYNDROME, PE, HYPOALBUMINEMIA

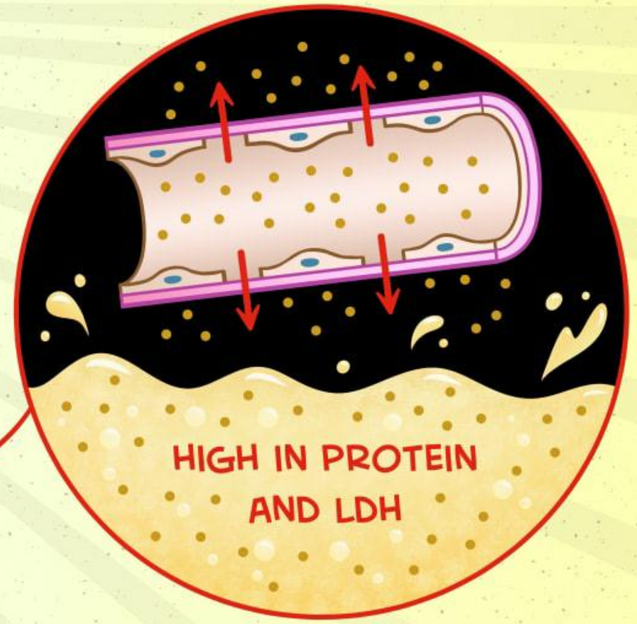




EXUDATIVE

OCCURS DUE TO INFLAMMATION AND INCREASED CAPILLARY PERMEABILITY

E.G., PNEUMONIA, CANCER, TB, VIRAL INFECTION, PE, AUTOIMMUNE



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Classifying Pleural Effusions

- A pleural effusion is an accumulation of fluid within the pleural space
- Determining the underlying cause is facilitated by thoracentesis and pleural fluid analysis
- The pleural fluid may be classified as a transudate or an exudate, depending on the etiology
- Transudates occur secondary to conditions which cause an increase in the pulmonary capillary hydrostatic pressure or a decrease in the capillary oncotic pressure
    - Leads to accumulation of protein poor pleural fluid
    - Common causes include: CHF, nephrotic syndrome, cirrhosis, hypoalbuminemia, pulmonary embolism
- Exudates occur secondary to conditions which cause inflammation or increased pleural vascular permeability
    - Leads to accumulation of protein rich pleural fluid and cells
    - Common causes include: pneumonia, cancer, tuberculosis, pulmonary embolism
- According to Light's criteria, if at least one of the following criteria is present, then the fluid is determined to be an exudate:
    - Pleural fluid protein to serum protein ratio greater than 0.5
    - Pleural fluid LDH to serum LDH ratio greater than 0.6
    - Pleural fluid LDH greater than two-thirds the upper limit for normal serum LDH

Presentation

- Often asymptomatic, but can present with dyspnea, pleuritic chest pain, and cough
- Physical examination may demonstrate decreased breath sounds on the side of the effusion, dullness to percussion, and decreased tactile fremitus

Imaging

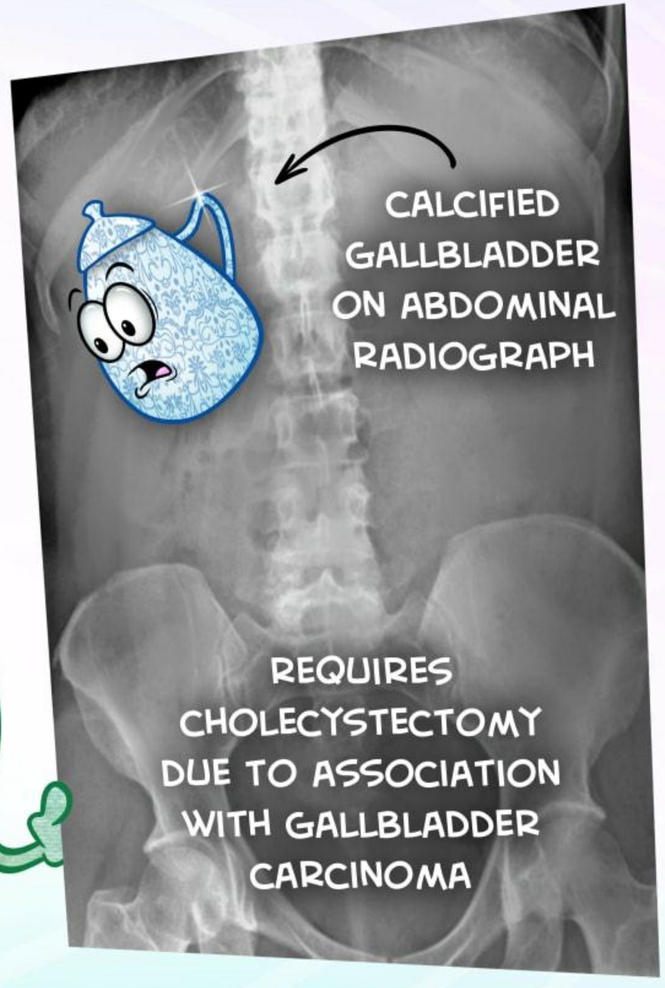
- Chest x-ray: blunting of costophrenic angles; free-flowing effusions will result in layering of fluid on the decubitus view
- Chest CT sometimes used for further evaluation

Treatment

- Treat underlying cause
- Thoracentesis is diagnostic and therapeutic
- Pleurodesis or indwelling catheter for recurrent/malignant effusions

PORCELAIN GALLBLADDER

CHRONIC CHOLELITHIASIS AND CHOLECYSTITIS CAUSES CALCIFIED SCARRING


CALCIFIED GALLBLADDER ON ABDOMINAL RADIOGRAPH
REQUIRES CHOLECYSTECTOMY DUE TO ASSOCIATION WITH GALLBLADDER CARCINOMA

COURVOISIER'S GALLBLADDER

ENLARGED, PALPABLE NONTENDER GALLBLADDER WITH JAUNDICE

ASSOCIATED WITH CANCER OF THE HEAD OF THE PANCREAS

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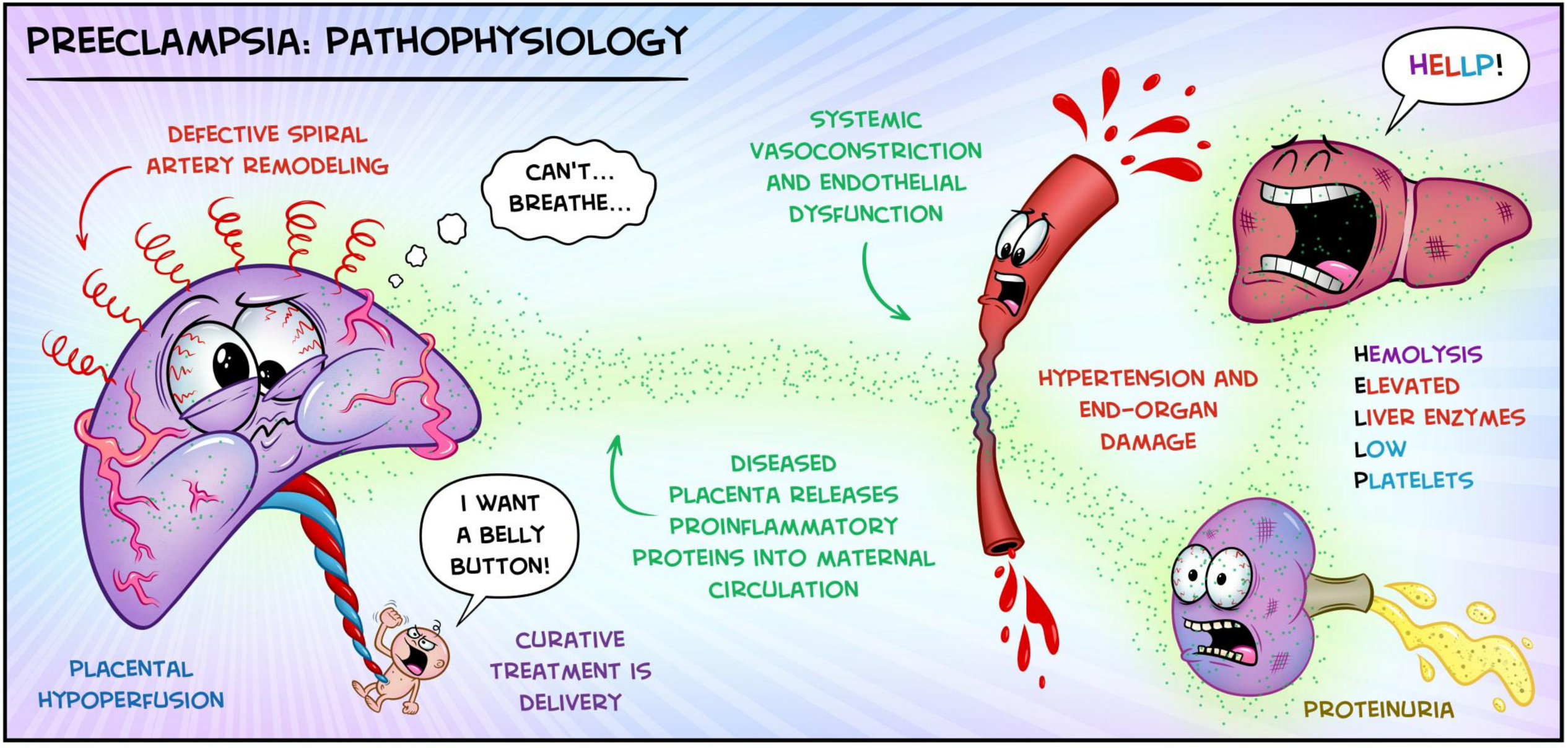
- Porcelain gallbladder: calcified gallbladder on abdominal radiograph due to scarring caused by chronic cholelithiasis/cholecystitis.
    - Requires cholecystectomy due to association with gallbladder carcinoma.
- Courvoisier's gallbladder: enlarged, palpable nontender gallbladder with jaundice.
    - Associated with cancer of the head of the pancreas.

PREECLAMPSIA: PATHOPHYSIOLOGY



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General Features

- Preeclampsia is classically defined as the new onset of hypertension and proteinuria after 20 weeks of gestation.
    - New onset hypertension with significant end-organ dysfunction (with or without proteinuria) after 20 weeks of gestation also satisfies the diagnosis of preeclampsia.
        - Severe headache, epigastric or right upper quadrant pain, visual disturbances, thrombocytopenia, impaired liver function, renal insufficiency, pulmonary edema
        - HELLP syndrome: Hemolysis, Elevated Liver enzymes, Low Platelets
    - Edema may or may not be present.
- Eclampsia = preeclampsia + seizures
- Risk factors include nulliparity, past history of preeclampsia, preexisting hypertension or renal disease, autoimmune disease, very young or advanced maternal age, diabetes, and obesity.
- Curative treatment is delivery

Pathophysiology

- Genetic and immunological factors
- Defective spiral artery remodeling during pregnancy
    - During a normal pregnancy trophoblast cells invade the myometrial segment of the spiral arteries to cause transformation and dilation of the arteries.
    - In preeclampsia there is insufficient trophoblast cell invasion, which causes the spiral arteries to remain narrow and leads to placental hypoperfusion.
- Diseased placenta releases proinflammatory proteins
    - Hypertension
    - Vasoconstriction
    - Endothelial cell dysfunction
        - Formation of thrombi
    - End-organ damage

OSTEOARTHRITIS

DEGENERATIVE DISEASE

MORNING STIFFNESS LASTING LESS THAN 30 MINUTES

HEBERDEN'S NODES

CARTILAGE LOSS

ASYMMETRICAL

INFLAMED SYNOVIUM

SYMMETRICAL

RHEUMATOID ARTHRITIS

AUTOIMMUNE DISEASE

MORNING STIFFNESS LASTING MORE THAN 30 MINUTES

EXTRA-ARTICULAR INVOLVEMENT

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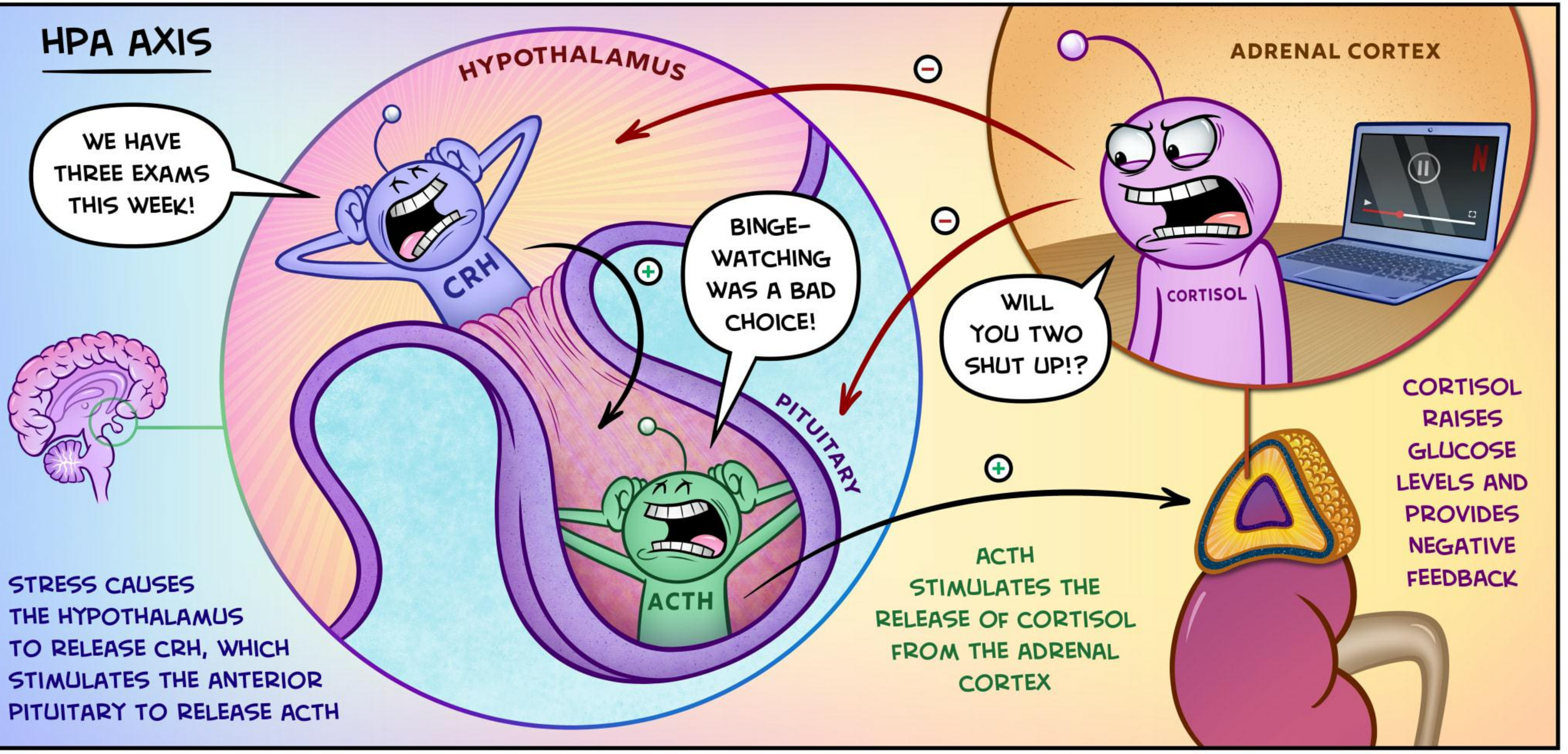
Osteoarthritis (OA)	Rheumatoid Arthritis (RA)
Degenerative disease that causes articular cartilage loss and joint space narrowing.	Autoimmune disease that causes inflammatory synovitis.
Heberden's nodes are bony prominences located at the distal interphalangeal (DIP) joints. Bouchard's nodes are located at the proximal interphalangeal (PIP) joints.	Heberden's nodes are absent. Bouchard's nodes are not common, but may be present.
Typically affects joint asymmetrically.	Typically affects joints symmetrically.
Usually negative/normal lab findings.	Positive rheumatoid factor, anti-CCP antibody, and elevated ESR and CRP.
Radiograph: subchondral sclerosis, osteophytes, cysts, joint space narrowing, subluxation.	Radiograph: periarticular osteopenia, bone erosions, joint space narrowing, subluxation.

HPA AXIS


HYPOTHALAMUS
WE HAVE THREE EXAMS THIS WEEK!
CRH
BINGE-WATCHING WAS A BAD CHOICE!
PITUITARY
ACTH
ADRENAL CORTEX
WILL YOU TWO SHUT UP!?
CORTISOL
STRESS CAUSES THE HYPOTHALAMUS TO RELEASE CRH, WHICH STIMULATES THE ANTERIOR PITUITARY TO RELEASE ACTH
ACTH STIMULATES THE RELEASE OF CORTISOL FROM THE ADRENAL CORTEX
CORTISOL RAISES GLUCOSE LEVELS AND PROVIDES NEGATIVE FEEDBACK

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The Adrenal Glands

- The adrenal glands are triangular shaped organs that sit on top of the kidneys.
- Each adrenal gland is composed of an outer portion called the cortex, and an inner portion called the medulla.
    - The adrenal cortex secretes mineralocorticoids, glucocorticoids, and androgens.
        - The cortex is further divided into three layers, or zones: glomerulosa (outer layer), fasciculata (middle layer), and reticularis (inner layer).
    - The innermost portion of the adrenal gland, the adrenal medulla, secretes catecholamines and is under the direct influence of the sympathetic nervous system.
        - Catecholamines: epinephrine and norepinephrine

The Adrenal Cortex (G-F-R)

Zona	Position	Hormone Class	Hormones	Deeper = Sweeter
Glomerulosa	Outer layer	Mineralocorticoid	Aldosterone	Salt
Fasciculata	Middle layer	Glucocorticoid	Cortisol	Sugar
Reticularis	Inner layer	Androgen	DHEA, DHEA-S, androstenedione	Sex

Hypothalamic-pituitary-adrenal Axis

- The hypothalamic-pituitary-adrenal (HPA) axis is a major neuroendocrine system that involves the hypothalamus, pituitary gland, and the adrenal glands.
- Corticotropin-releasing hormone (CRH)
    - Stress response
    - Released from hypothalamus
    - Stimulates the release of adrenocorticotropic hormone (ACTH)
- ACTH
    - Released from anterior pituitary
    - Stimulates the release of cortisol
- Cortisol
    - Released from adrenal gland (zona fasciculata of adrenal cortex)
    - Provides negative feedback, acting on the hypothalamus and pituitary to suppress CRH and ACTH production

MONTEGGIA

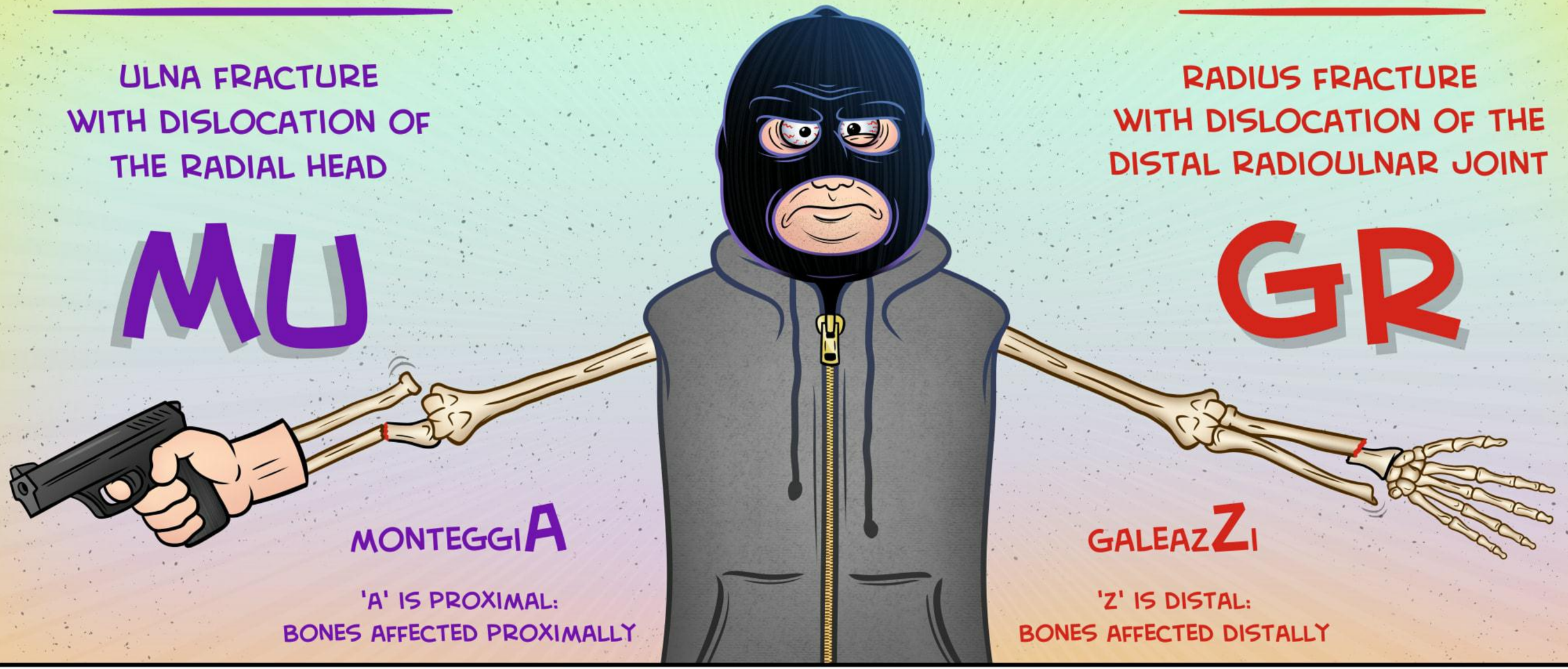
ULNA FRACTURE
WITH DISLOCATION OF
THE RADIAL HEAD

MU

MONTEGGI**A**

'A' IS PROXIMAL:
BONES AFFECTED PROXIMALLY

"MUGGER"



GALEAZZI

RADIUS FRACTURE
WITH DISLOCATION OF THE
DISTAL RADIOULNAR JOINT

GR

GALEAZ**Z**I

'Z' IS DISTAL:
BONES AFFECTED DISTALLY

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Definitions

- A Monteggia fracture is a fracture of the proximal ulna with dislocation of the radial head.
    - There are four types, based on the direction that the radial head is displaced (Bado classification).
- A Galeazzi fracture is a fracture of the distal radius with dislocation of the distal radioulnar joint (DRUJ).

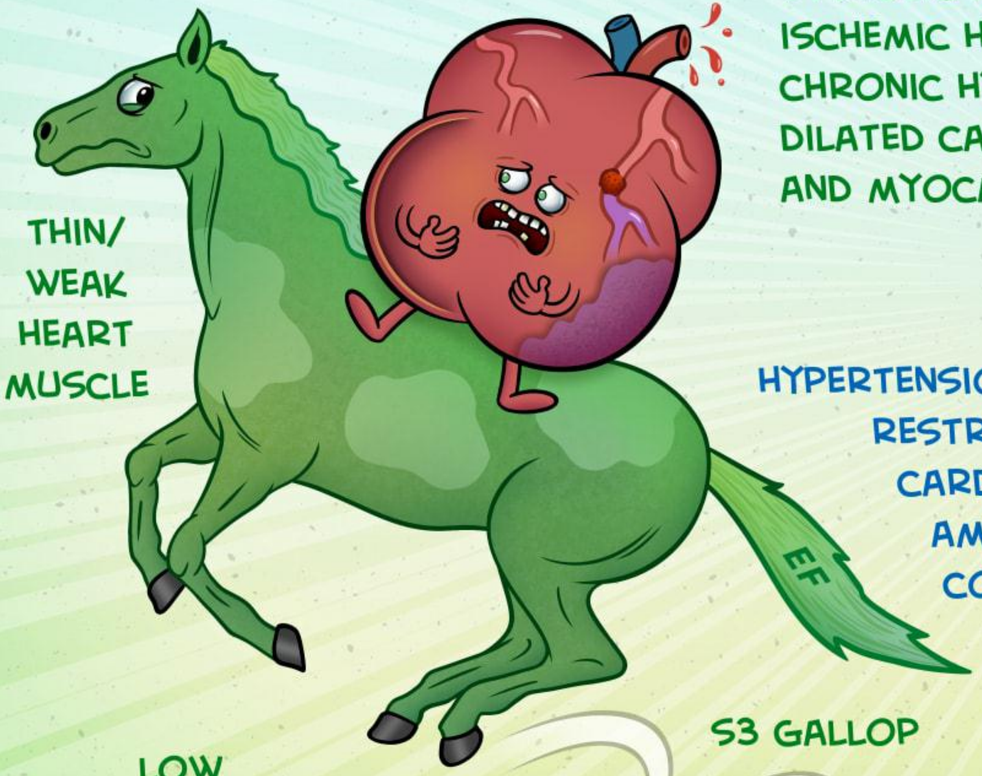
Fracture-dislocation Mnemonic

- MUGR (pronounced "MUGGER"): identifies the fractured bone.
    - MU: Monteggia Ulna
    - GR: Galeazzi Radius
- Use "A to Z" to remember the location of the fracture-dislocation.
    - 'A' is proximal (Monteggi**A**)
        - Radial head dislocation and proximal ulna fracture
    - 'Z' is distal (Galeaz**Z**i)
        - Distal radioulnar joint dislocation and distal radius fracture

SYSTOLIC DYSFUNCTION

IMPAIRED CONTRACTILITY

THIN/ WEAK HEART MUSCLE



LOW EJECTION FRACTION

S3 GALLOP

SYS-TOL-IC

SYSTOLIC AND DIASTOLIC DYSFUNCTION CAN APPEAR IN COMBINATION

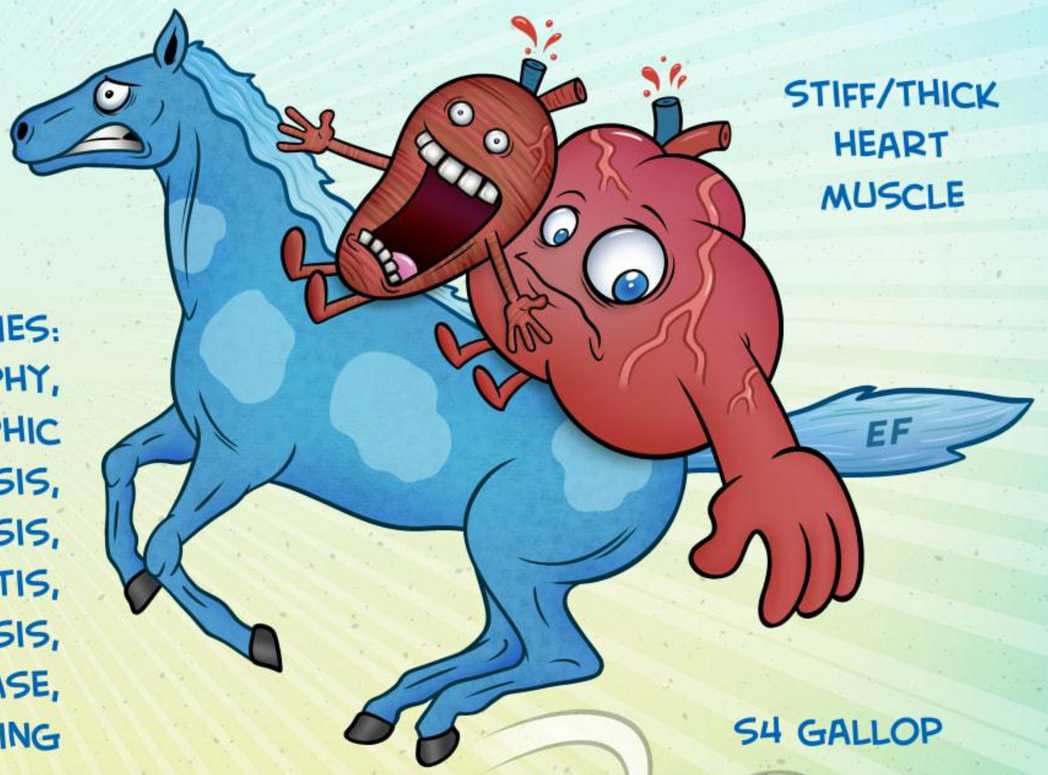
SYSTOLIC ETIOLOGIES: ISCHEMIC HEART DISEASE, CHRONIC HYPERTENSION, DILATED CARDIOMYOPATHY, AND MYOCARDITIS

DIASTOLIC ETIOLOGIES: HYPERTENSION WITH LV HYPERTROPHY, RESTRICTIVE AND HYPERTROPHIC CARDIOMYOPATHIES, FIBROSIS, AMYLOIDOSIS, SARCOIDOSIS, CONSTRICTIVE PERICARDITIS, HEMOCHROMATOSIS, VALVULAR DISEASE, AND AGING

DIASTOLIC DYSFUNCTION

IMPAIRED FILLING/RELAXATION

STIFF/THICK HEART MUSCLE



S4 GALLOP

NORMAL EJECTION FRACTION

DI-AS-TOL-IC

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General Considerations

- Heart failure is defined as the inability of the heart to provide sufficient output to meet the metabolic demands of the body.
- Heart failure may be classified by symptom severity (NYHA classification) and by stage of evolution (ACC/AHA classification).
- Heart failure may also be classified by type of dysfunction (systolic vs. diastolic) and by the side of the heart affected (right vs. left).
    - The terms used to classify heart failure are not mutually exclusive – the conditions may be present at the same time (e.g. left heart failure with diastolic dysfunction, right heart failure with systolic dysfunction).
    - There may be overlap within categories (e.g. heart failure with systolic and diastolic dysfunction, biventricular heart failure).
    - The most common cause of right-sided heart failure is left-sided heart failure.

NEW YORK HEART ASSOCIATION (NYHA)
HEART FAILURE CLASSIFICATION

CLASS I

NO LIMITATION OF PHYSICAL ACTIVITY; ORDINARY PHYSICAL ACTIVITY DOES NOT CAUSE SYMPTOMS

CLASS II

SLIGHT LIMITATION OF PHYSICAL ACTIVITY; COMFORTABLE AT REST; ORDINARY PHYSICAL ACTIVITY CAUSES SYMPTOMS

CLASS III

MARKED LIMITATION OF PHYSICAL ACTIVITY; COMFORTABLE AT REST, BUT LESS THAN ORDINARY ACTIVITY CAUSES SYMPTOMS

CLASS IV

SEVERE LIMITATION AND DISCOMFORT WITH ANY PHYSICAL ACTIVITY; SYMPTOMS PRESENT EVEN AT REST

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Functional Classification Systems

- The New York Heart Association (NYHA) functional classification is based on the amount of activity needed to elicit symptoms from the patient.
- The American College of Cardiology/American Heart Association (ACC/AHA) classification describes the progressive stages in the development of heart failure.
- Signs and symptoms associated with heart failure include fatigue, exertional and resting dyspnea, orthopnea, peripheral edema, and increased jugulovenous distention (JVD), among others.
- Examples of conditions that cause low output heart failure include coronary artery disease, hypertension, cardiomyopathy, pericardial disease, arrhythmias, and valvular disease.
- Examples of conditions that cause high output heart failure include severe anemia, thyrotoxicosis, Paget's disease of bone, sepsis, systemic arteriovenous shunts, and beriberi.

NYHA Functional Classification of Heart Failure

NYHA Class	Description
I	No limitation of physical activity; ordinary physical activity does not cause symptoms
II	Slight limitation of physical activity; comfortable at rest; ordinary physical activity causes symptoms
III	Marked limitation of physical activity; comfortable at rest, but less than ordinary activity causes symptoms
IV	Severe limitation and discomfort with any physical activity; symptoms present even at rest

ACC/AHA Stages of Heart Failure

Stage	Description
A	At high risk for the development of heart failure, but without any structural heart disease, functional heart abnormality, or symptoms of heart failure
B	Structural heart disease, but without signs or symptoms of heart failure
C	Structural heart disease with past or current symptoms of heart failure
D	Advanced heart disease with refractory heart failure requiring specialized interventions

HEART FAILURE (HF): PATHOPHYSIOLOGY

THE INABILITY OF THE HEART TO PROVIDE SUFFICIENT OUTPUT TO MEET THE DEMANDS OF THE BODY

A VARIETY OF DISORDERS CAN LEAD TO LOW OUTPUT OR HIGH OUTPUT FAILURE

INCREASED SYMPATHETIC NERVOUS SYSTEM ACTIVITY

HEY... SHOW THE POOR HEART SOME SYMPATHY.

PUT A CORK IN IT. WE'RE RELEASING HORMONES.

NEURO-HORMONAL RESPONSES WORSEN HF

INCREASED AFTERLOAD

RAAS

ADH SECRETION FROM THE BRAIN

AORTA

CONGESTION

WE NEED BLOOD FLOW! TRY HARDER!

SODIUM AND WATER RETENTION

PULMONARY AND SYSTEMIC VENOUS CONGESTION

HELP...

WE'RE GETTING BACKED UP. DO YOUR JOB!

CARDIAC DILATION AND HYPERTROPHY

INCREASED PRELOAD

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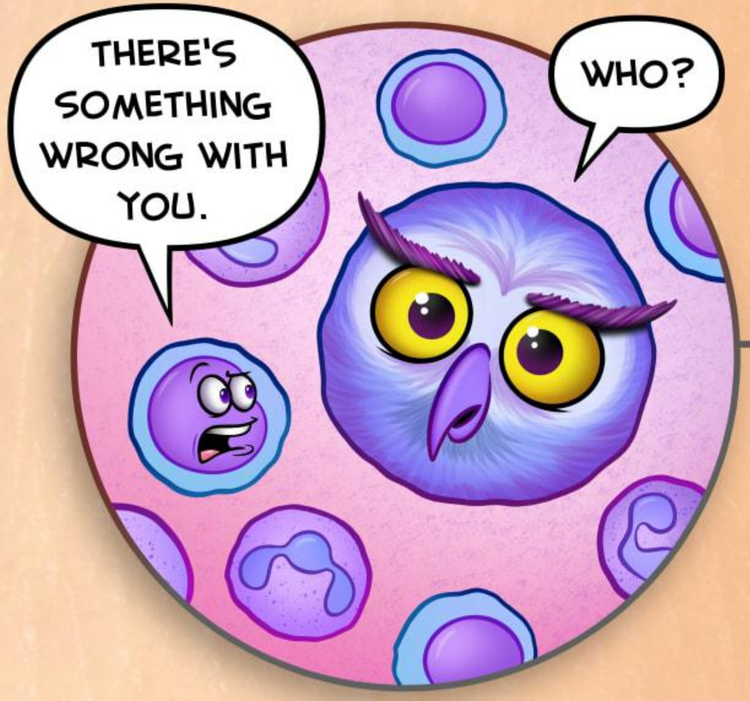
General Features and Pathophysiology

- Heart failure (HF) is defined as the inability of the heart to provide sufficient output to meet the metabolic demands of the body.
    - Most often a chronic condition, but may also be acute.
- Heart failure leads to tissue hypoperfusion, as well as pulmonary and systemic venous congestion.
- There are many possible etiologies. Low output failure occurs with conditions associated with impaired cardiac contractility, impaired cardiac filling, excess afterload, or arrhythmias. High output failure describes a condition in which the signs and symptoms of heart failure occur in the setting of increased cardiac output, reduced systemic vascular resistance, and increased metabolic demand.
    - Examples of conditions that cause low output heart failure include coronary artery disease, hypertension, cardiomyopathy, pericardial disease, arrhythmias, and valvular disease.
    - Examples of conditions that cause high output heart failure include severe anemia, thyrotoxicosis, Paget's disease of bone, sepsis, systemic arteriovenous shunts, and beriberi.
- Compensatory mechanisms develop to improve cardiac output, but in time these responses only serve to exacerbate the underlying cardiac problem. Examples of compensatory mechanisms include:
    - Neurohormonal activation
        - Increased sympathetic nervous system activity causes increased myocardial contractility and peripheral vasoconstriction.
        - Activation of the renin-angiotensin-aldosterone system leads to sodium and water retention.
        - Vasopressin/ADH secretion causes peripheral vasoconstriction and water retention.
        - Neurohormonal activation helps maintain systemic arterial pressures, but also contributes to an increased workload on the heart.
    - Cardiac dilation and hypertrophy

HODGKIN LYMPHOMA

CLONAL B-CELL MALIGNANCY THAT DEVELOPS WITHIN THE LYMPHATIC SYSTEM



THE MALIGNANT REED-STERNBERG CELL TYPICALLY HAS A BILOBED NUCLEUS THAT GIVES AN "OWL'S EYES" APPEARANCE

DIAGNOSIS: EXCISIONAL LYMPH NODE BIOPSY

SPREADS IN AN ORDERLY FASHION TO ADJACENT NODES

IT'S SPREADING TO YOU NEXT!

WHO?

PAINLESS LYMPHADENOPATHY, CONSTITUTIONAL "B" SYMPTOMS (FEVER, NIGHT SWEATS, WEIGHT LOSS), PRURITUS, HEPATOSPLENOMEGALY

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Hodgkin lymphoma (HL) is a clonal B-cell malignancy that resides predominantly in the lymphatic system. HL tends to develop within a single lymph node region and spreads in an orderly fashion to adjacent lymph nodes. A defining characteristic of HL is the presence of the Reed-Sternberg cell – a large cell with a bilobed nucleus and prominent nucleoli, giving the appearance of "owl's eyes."

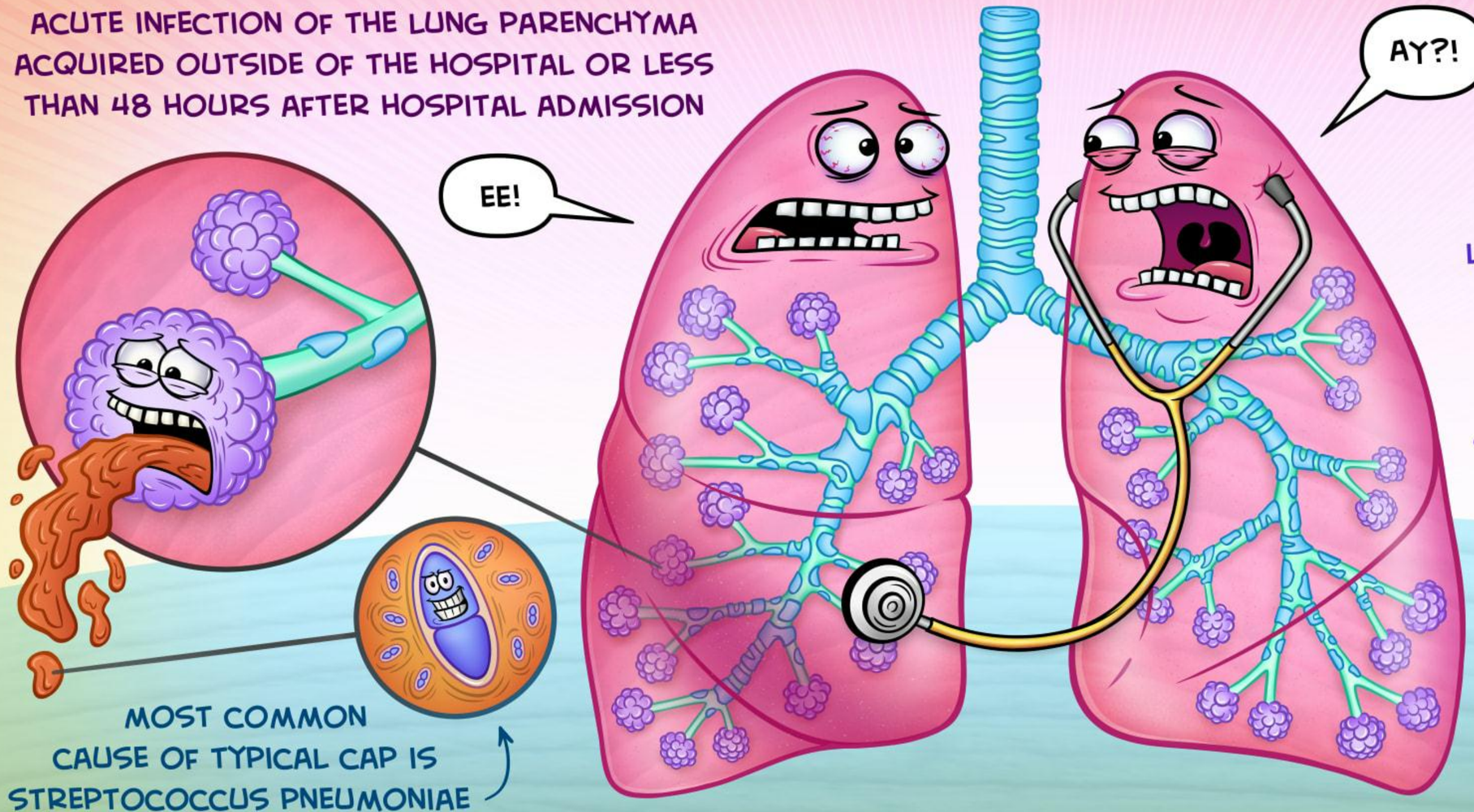
There is a bimodal age distribution, peaking among patients in their 20s and in those greater than 50 years of age. HL commonly presents with painless lymphadenopathy (cervical, supraclavicular, and mediastinal). Other clinical manifestations include constitutional "B" symptoms (fever, drenching night sweats, and weight loss), pruritus, and hepatosplenomegaly. A suggestive symptom is pain in the affected lymph node after alcohol consumption. HL is associated with Epstein-Barr virus infection.

The diagnosis is made by excisional lymph node biopsy. Nodular sclerosing is the most common histologic type of HL. The staging evaluation includes CT scans (chest, abdomen, pelvis, and sometimes neck), PET scans, and sometimes bone marrow biopsy if advanced disease is suspected. The Ann Arbor system is used to stage HL with "A" and "B" modifiers. Stage A represents HL without symptoms (think: A = Asymptomatic). Stage B represents HL with constitutional symptoms, which is associated with a poorer prognosis (think: B = Bad). Treatment involves radiation therapy and/or combination chemotherapy.

TYPICAL COMMUNITY ACQUIRED PNEUMONIA

ACUTE INFECTION OF THE LUNG PARENCHYMA ACQUIRED OUTSIDE OF THE HOSPITAL OR LESS THAN 48 HOURS AFTER HOSPITAL ADMISSION

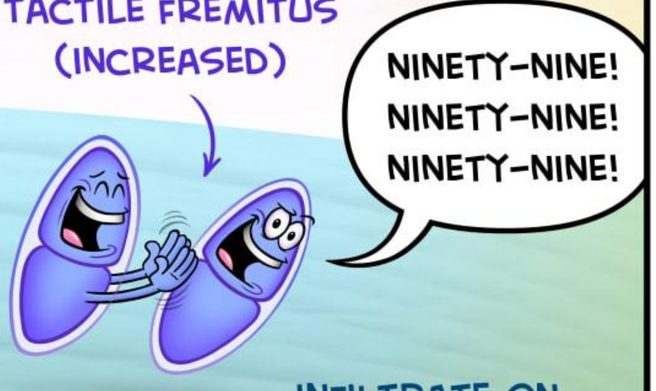
FEVER, COUGH, SPUTUM PRODUCTION, RIGORS, PLEURITIC CHEST PAIN, DYSPNEA, TACHYCARDIA



BRONCHIAL BREATH SOUNDS AND CRACKLES ON AUSCULTATION

SPECIAL FINDINGS DUE TO LOBAR CONSOLIDATION INCLUDE:

- EGOPHONY (E TO A)
- WHISPERED PECTORILOQUY
- TACTILE FREMITUS (INCREASED)



INFILTRATE ON CHEST RADIOGRAPH

MOST COMMON CAUSE OF TYPICAL CAP IS STREPTOCOCCUS PNEUMONIAE

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Community acquired pneumonia (CAP) is an acute infection of the lung parenchyma acquired outside of the hospital or less than 48 hours after hospital admission. CAP is classified into typical and atypical subtypes, differentiated by their presentation and causative pathogens. This illustration focuses on the classic features of typical CAP.

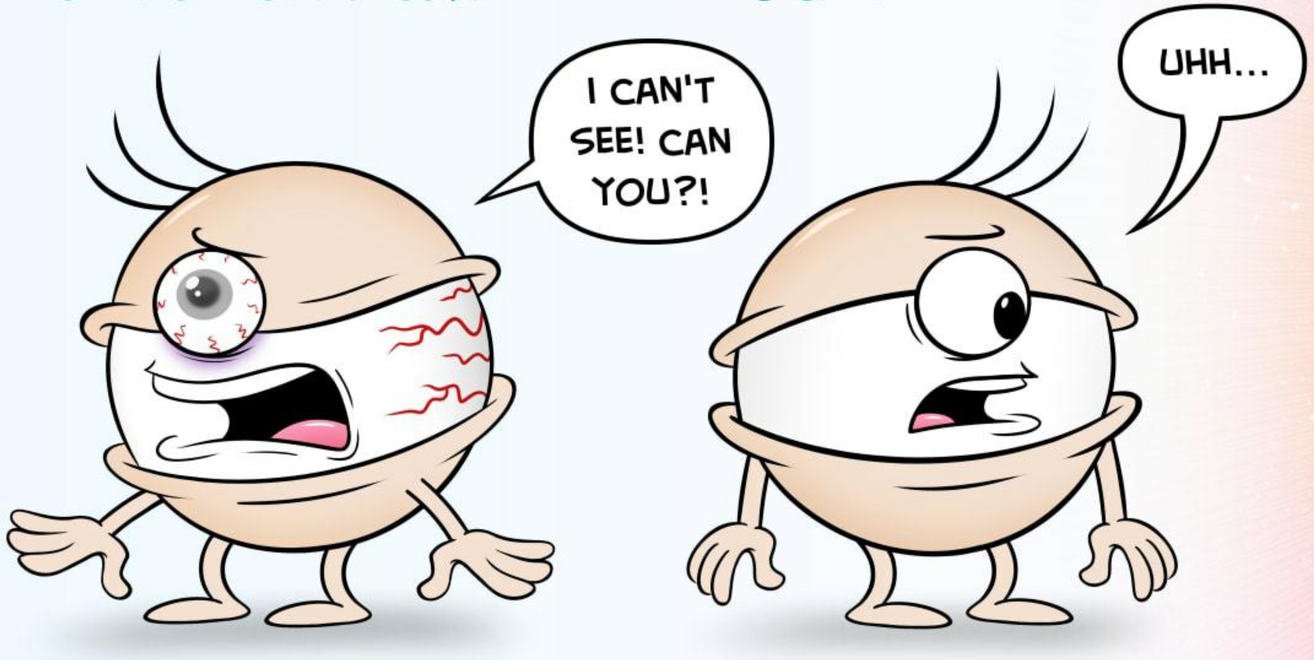
The most common cause of typical CAP is *Streptococcus pneumoniae*. It is an encapsulated, gram-positive, lancet-shaped diplococcus bacterium. Other common causative pathogens include *Haemophilus influenzae*, *Moraxella catarrhalis*, gram-negative bacilli (e.g. *Klebsiella*), and *Staphylococcus aureus*. Common viral agents include influenza viruses, respiratory syncytial virus (RSV), adenovirus, and parainfluenza viruses.

Typical CAP is characterized by the acute onset of fever, cough, sputum production, rigors, pleuritic chest pain, dyspnea, and tachycardia. *Streptococcus pneumoniae* infection is classically associated with the production of rust-colored sputum. Bronchial breath sounds and crackles may be heard on auscultation. Special findings due to lobar consolidation include egophony (E to A), whispered pectoriloquy, and increased tactile fremitus. CAP in the setting of a pleural effusion may demonstrate decreased tactile fremitus and dullness to percussion. Chest radiography is important in establishing the diagnosis, which may reveal lobar consolidation, patchy airspace opacities, or interstitial opacities. Treatment involves empiric antibiotics or organism-specific antibiotics if the pathogen is identified.

VISUAL RELEASE HALLUCINATIONS

AKA CHARLES BONNET SYNDROME (CBS)

VISUAL HALLUCINATIONS THAT OCCUR DUE TO VISION LOSS FROM ANY UNDERLYING CAUSE



EXAMPLES OF UNDERLYING CAUSES:
MACULAR DEGENERATION, GLAUCOMA, DIABETIC RETINOPATHY, OPTIC NEURITIS, SURGERY, STROKE

HALLUCINATIONS MAY BE SIMPLE OR COMPLEX

MORE COMMON IN ELDERLY PATIENTS

PATIENTS UNDERSTAND THAT THE HALLUCINATIONS ARE NOT REAL

THE DIAGNOSIS IS MADE IN THE ABSENCE OF PSYCHIATRIC DISORDERS OR OTHER CAUSES OF HALLUCINATIONS

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Visual release hallucinations, also known as Charles Bonnet syndrome (CBS), refers to symptoms of visual hallucinations that occur due to vision loss from any underlying cause. The underlying condition may affect the eye, optic nerve, or the brain. Examples of underlying causes include macular degeneration, glaucoma, diabetic retinopathy, optic neuritis, surgery, and stroke.

CBS is thought to occur due to the interruption or destruction of the afferent connections of nerve cells in the visual system leading to disinhibition of the visual cortex. The subsequent spontaneous firing of neurons causes hallucinations.

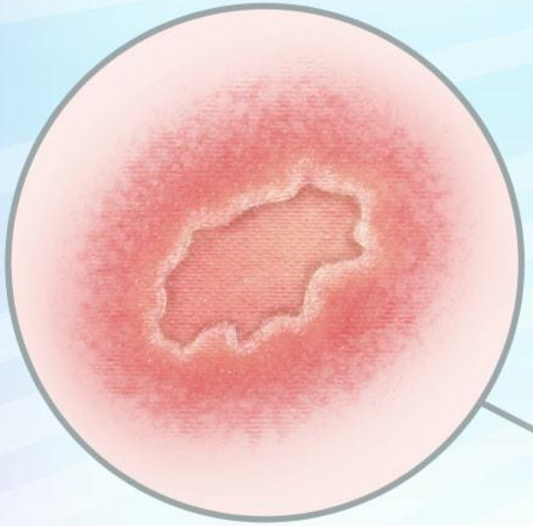
Patients with CBS have insight into the unreal nature of their hallucinations. It is not a symptom of psychiatric illness or any other cause of dementia. CBS can occur in all age groups, but is more common in elderly patients. Patients may experience simple hallucinations, such as flashes of light or shapes, or they can be complex hallucinations, such as images of people, animals, landscapes, or cartoons. The hallucinations are commonly pleasant, but may cause anxiety. The duration of the hallucinations generally depends on the underlying cause. In some cases they may only last days to weeks, while in other cases the hallucinations may persist over several years.

The diagnosis of CBS is made in patients with vision loss in the absence of psychiatric disorders or other causes of hallucinations.

CBS is managed by treating the underlying condition causing the visual deficit, and providing reassurance to patients of their sanity. In some cases anticonvulsant or antipsychotic therapy may provide relief from CBS.

PITYRIASIS ROSEA

AN ACUTE ERUPTION OF OVAL, SCALY PAPULES AND PLAQUES THAT BEGINS WITH A SINGLE HERALD PATCH



1-2 WEEKS LATER THE ERUPTION APPEARS ON THE TORSO IN A CHRISTMAS TREE PATTERN

THIS IS A PITY.

A VIRAL ETIOLOGY IS PROBABLE (HHV-6 AND HHV-7)

SUNLIGHT OR UVB PHOTOTHERAPY MAY HELP THE LESIONS DISAPPEAR FASTER

MAINLY SEEN IN TEENAGE AND YOUNG ADULT FEMALES

CONDITION RESOLVES SPONTANEOUSLY IN ABOUT 6 WEEKS

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Pityriasis rosea is an acute, self-limited eruption of oval, scaly papules and plaques with a distinctive morphology. Females are affected more often than males, and it most common in teenagers and young adults. Pityriasis rosea peaks in the spring and fall seasons. The most probable cause has been attributed to reactivation of human herpesvirus (HHV-6 and HHV-7).

Pirytiasis rosea begins with a single lesion known as a "herald" patch, usually located on the trunk. This initial lesion is an erythematous, oval plaque with a collarette of scale inside the border. Approximately 1-2 weeks after the appearance of the herald patch, a characteristic rash appears on the torso that follows the lines of cleavage in a "Christmas tree" pattern. Pityrias rosea may be accompanied by pruritis.

The condition resolves spontaneously in about 6 weeks often requires no treatment. Sunlight or UVB phototherapy may help the lesions disappear faster if started during the first week of eruption. Lotions, oral antihistamines, and/or topical glucocorticoids may be given for itching.

CLOSTRIDIUM DIFFICILE INFECTION

C. DIFFICILE CONTAINS ENDOSPORES THAT CAN SURVIVE THE ACIDITY OF THE STOMACH AND REACH THE LARGE INTESTINE

OOPS.

CLINDAMYCIN

C. DIFFICILE FLOURISHES WITHIN THE COLON

FEVER, CRAMPY ABDOMINAL PAIN, DIARRHEA

MOST COMMON INFECTIOUS CAUSE OF NOSOCOMIAL DIARRHEA

DIARRHEA

TOXINS A & B CAUSE MUCOSAL DAMAGE

PSEUDOMEMBRANOUS COLITIS: YELLOWISH PLAQUES FORM OVER DAMAGED EPITHELIUM

THE NORMAL GUT FLORA IS ALTERED BY BROAD-SPECTRUM ANTIBIOTICS, MOST NOTABLY CLINDAMYCIN, CEPHALOSPORINS, AMPICILLIN, AMOXICILLIN, AND FLUOROQUINOLONES

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Clostridium difficile is a gram-positive anaerobic bacillus which secretes toxins (A and B) that cause diarrhea and pseudomembranous colitis. *C. difficile* is the most common infectious cause of nosocomial diarrhea. Transmission of *C. difficile* can occur from contact with contaminated humans and fomites. The organism contains endospores that can survive the acidity of the stomach and reach the large intestine.

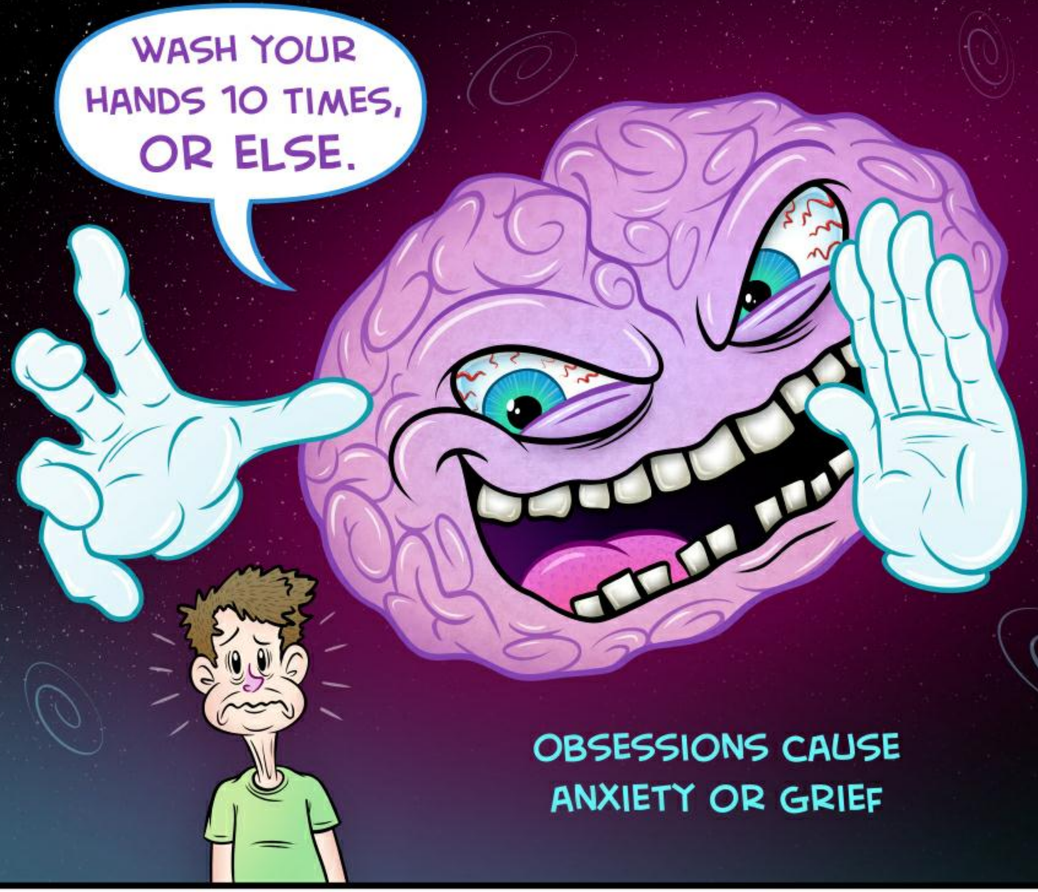
C. difficile colonizes the intestinal tract after the normal gut flora has been altered by antibiotic therapy. It can flourish after any antibiotic therapy, but most notably occurs with broad-spectrum antibiotics such as clindamycin, cephalosporins, ampicillin, amoxicillin, and fluoroquinolones. Pseudomembranous colitis is an inflammatory bowel disorder in which yellowish plaques form over the damaged intestinal mucosa. The plaque is an exudate that resembles a membrane, hence the term "pseudomembranous."

The signs and symptoms include fever, crampy abdominal pain, and diarrhea. Progression of the condition can lead to leukocytosis, dehydration, and toxic megacolon. The diagnosis is confirmed with a stool assay for *C. difficile* toxin. Pseudomembranes can be seen on sigmoidoscopy.

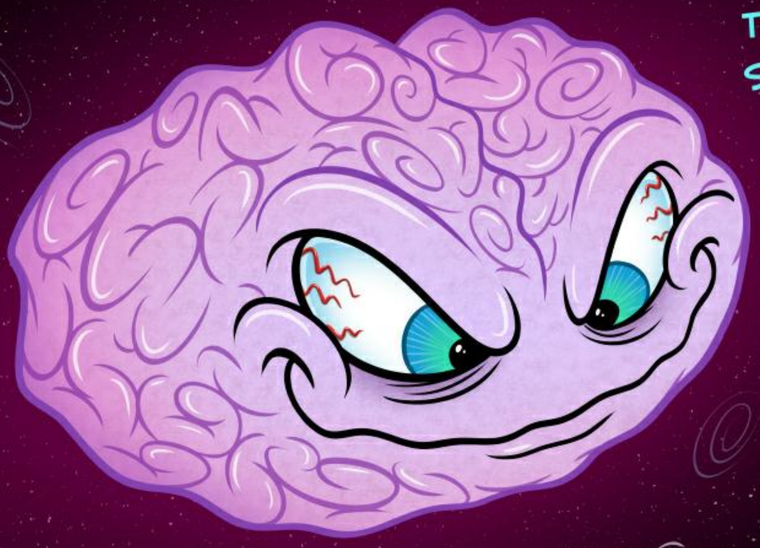
Treatment includes discontinuing the causative agent. Oral metronidazole is the treatment of choice in patients that do not respond to conservative measures. Alternatively, very severe cases may require oral vancomycin. Rarely, cases complicated by toxic megacolon or perforation may require total colectomy.

OBSESSIVE-COMPULSIVE DISORDER

RECURRENT, INTRUSIVE, UNDESIRED THOUGHTS (OBSESSIONS) AND/OR UNCONTROLLABLE REPETITIVE ACTS (COMPULSIONS)



OBSESSIONS CAUSE ANXIETY OR GRIEF



OBSESSIONS AND COMPULSIONS ARE TIME CONSUMING AND CAUSE SIGNIFICANT DISTRESS IN A PATIENT'S DAILY LIFE

TREATMENT OPTIONS: SSRIs (TITRATED UP), CLOMIPRAMINE, BEHAVIORAL THERAPY

COMPULSIONS CAN CONSIST OF PHYSICAL BEHAVIORS OR MENTAL ACTS, AND MAY TEMPORARILY RELIEVE ANXIETY

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Obsessive-compulsive disorder (OCD) is characterized by recurrent, intrusive, undesired thoughts (known as obsessions) and/or uncontrollable repetitive acts (known as compulsions). Obsessions and compulsions are time-consuming (> 1 hour in a day), and cause significant distress in a patient's daily life. The prevalence is 2-3%, with a mean onset in the second decade.

Obsessions cause grief and anxiety. These thoughts are unreasonable and are not worries about real-life problems. Carrying out compulsions may temporarily relieve the anxiety caused by obsessions. Compulsions can include physical acts (e.g. frequent hand-washing, repeatedly checking the stove or door lock, arranging objects in a specific order) or mental acts (e.g. counting, repeating certain phrases).

Treatment options include cognitive behavioral therapy and pharmacotherapy, and combining the two may be best. SSRIs may need to be titrated to higher doses than those used to treat other disorders. Clomipramine is also effective, but has many side effects. Antipsychotics may be helpful as a third-line agent in treatment-resistant cases.

THE MENSTRUAL CYCLE

OVARIAN CYCLE

LOW ESTROGEN AT THE END OF MENSES

E F ESTROGEN

FSH

ESTROGEN

LH

SMACK!

PROGESTERONE

CORPUS LUTEUM

CORPUS ALBICANS

MENSES

FOLLICULAR PHASE

OVULATION!

LUTEAL PHASE

UTERINE CYCLE

PROLIFERATIVE PHASE: INFLUENCED BY ESTROGEN, THE THICKNESS OF THE ENDOMETRIUM RAPIDLY INCREASES

SECRETORY PHASE: INFLUENCED BY PROGESTERONE, THE LINING BECOMES HIGHLY VASCULAR AND EDEMATOUS

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The menstrual cycle is coordinated by a series of hormones that regulate the growth of the endometrium, the development of an oocyte, release and possible implantation of an ovum, and if pregnancy does not occur, sloughing of the endometrium and menses to allow a repeat of the same process. The first day of the menstrual cycle is represented by the first day of menses. The menstrual cycle is then divided into the follicular and luteal phases of the ovarian cycle, which correspond with the proliferative and secretory phases of the uterine cycle.

The follicular phase is initiated by low estrogen levels at the end of menses. Follicle-stimulating hormone (FSH) from the anterior pituitary stimulates the follicles in the ovary to grow and produce estrogen. This stage roughly corresponds with the proliferative phase of the uterine cycle, in which the thickness of the endometrium rapidly increases. The dominant follicle (Graafian follicle) emerges by day 7 and as estrogen continues to increase it inhibits the release of FSH. Luteinizing hormone (LH) surges mid-cycle in response to peak amounts of estrogen from the mature follicle. One ovum is released 36 to 42 hours from the onset of the LH surge.

The luteal phase is so named because it centers on the activity of the corpus luteum, the remnant of the ovarian follicle, which secretes progesterone to ready the uterus for a possible pregnancy. This stage roughly corresponds with the secretory phase of the uterine cycle, in which the lining of the uterus becomes highly vascular and edematous. If the egg is not fertilized, the corpus luteum involutes and becomes the corpus albicans (fibrous scar tissue) and menstruation occurs.

PULMONARY HYPERTENSION

ELEVATED MEAN ARTERIAL PRESSURE ≥ 25 MM HG AT REST ASSESSED BY RIGHT HEART CATHETERIZATION

CATEGORIZED INTO FIVE GROUPS BY THE WORLD HEALTH ORGANIZATION (WHO)

GROUP 1: PULMONARY ARTERIAL HYPERTENSION (PAH)

PAH EXAMPLES: IDIOPATHIC, INHERITED, DRUG AND TOXIN INDUCED, CAUSED BY CONNECTIVE TISSUE DISEASE, HIV, SCHISTOSOMIASIS

HEART CAT(H)

ONLY GROUP 1 IS CALLED PULMONARY "ARTERIAL" HYPERTENSION, BUT ALL 5 GROUPS MAY BE REFERRED TO AS PULMONARY HYPERTENSION (PH)

GROUP 3: PH DUE TO LUNG DISEASE AND/OR HYPOXEMIA

GROUP 4: PH DUE CHRONIC THROMBOEMBOLISM

GROUP 2: PH DUE TO LEFT HEART DISEASE (MOST COMMON)

GROUP 5: PH WITH UNCLEAR MULTIFACTORIAL MECHANISMS

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Pulmonary hypertension (PH) refers to increased pressure in the pulmonary circulation. It develops when pulmonary vessels become constricted and/or obstructed, which can occur in a wide variety of conditions. The increase in pressure is measured by right catheterization, and is defined as a mean pulmonary arterial pressure ≥ 25 mm Hg at rest.

PH leads to right ventricular hypertrophy and enlargement as the ventricle pumps against increased resistance. Right-sided heart failure (cor pulmonale) can eventually develop. Signs and symptoms of PH include dyspnea on exertion, fatigue, substernal chest pain, syncope, hepatomegaly, neck vein distention, and leg edema. Narrow splitting of S2 with an accentuated pulmonic component (P2) may be heard on auscultation.

Echocardiography is a helpful initial test to estimate the mean arterial pressure and may also demonstrate enlargement of the right ventricle and atrium, with tricuspid regurgitation. Echocardiography also allows for an assessment of left ventricular function, valvular disease, and congenital heart disease. The mean arterial pressure is definitively diagnosed with right heart catheterization.

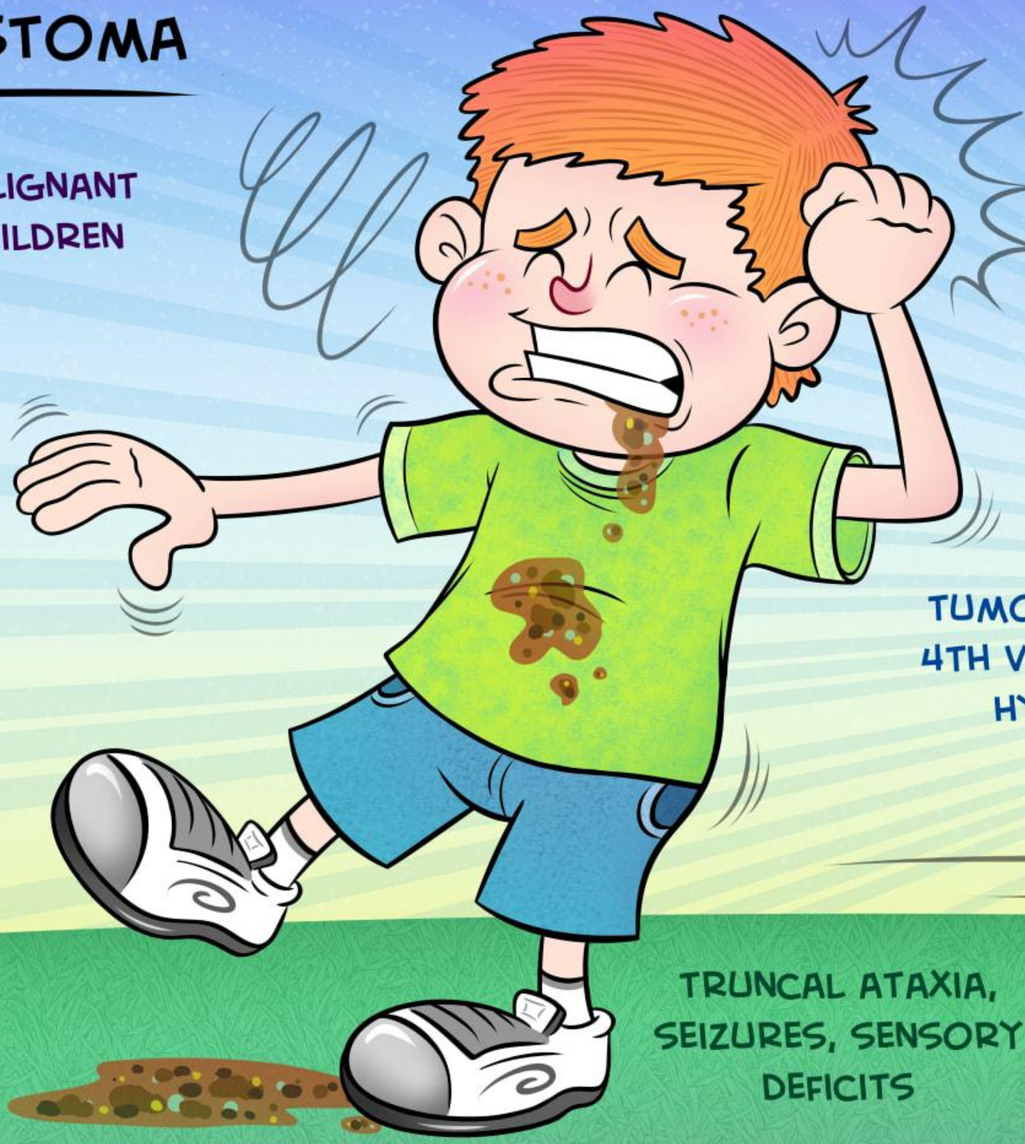
The World Health Organization (WHO) categorizes PH into 5 groups and the management of PH depends on the etiology. Possible underlying causes should be identified and treated. Treatment options include oxygen therapy, chronic anticoagulation, calcium channel blockers, prostacyclin (vasodilator), PDE5 inhibitors, and heart-lung transplantation in severe cases.

MEDULLOBLASTOMA

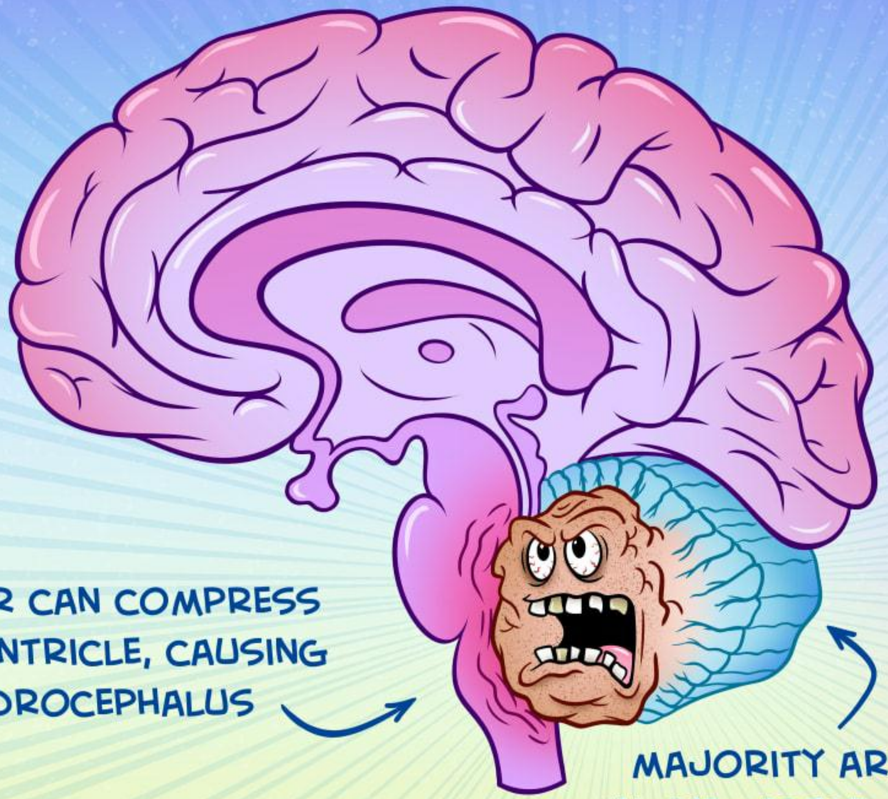
MOST COMMON MALIGNANT BRAIN TUMOR IN CHILDREN

HEADACHE, NAUSEA, VOMITING, DIZZINESS, VISUAL DISTURBANCES

IMAGING MODALITY OF CHOICE IS MRI



TRUNCAL ATAXIA, SEIZURES, SENSORY DEFICITS



TUMOR CAN COMPRESS 4TH VENTRICLE, CAUSING HYDROCEPHALUS

MAJORITY ARISE IN THE CEREBELLUM

TREATMENT USUALLY CONSISTS OF SURGERY, RADIATION, AND CHEMOTHERAPY

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Medulloblastoma is the most common type of malignant brain tumor in childhood. It is an aggressive solid tumor malignancy that develops in the posterior fossa, which contains the brain stem and cerebellum. Boys are affected more frequently than girls, with cases usually occurring between 5 and 9 years of age.

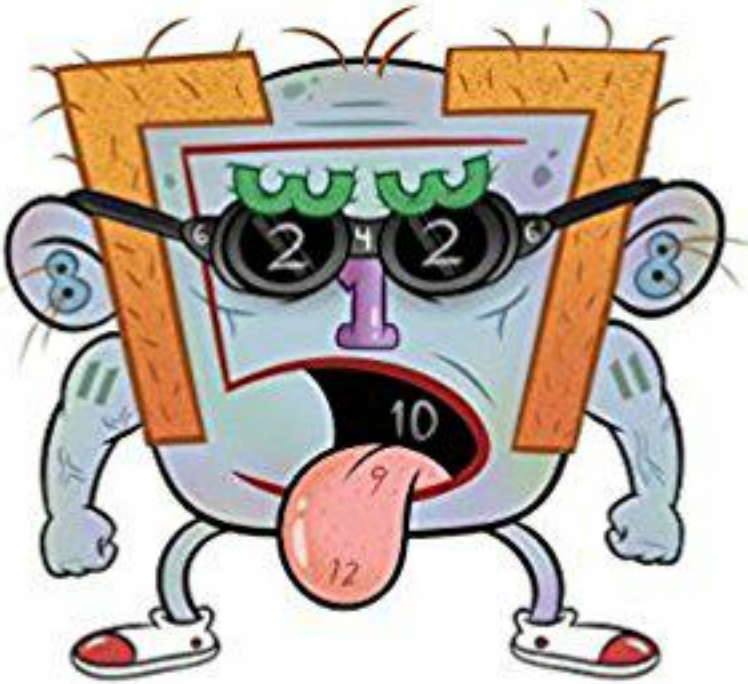
Signs and symptoms are related to cerebellar dysfunction and increased intracranial pressure due to blockage of the fourth ventricle. Patients commonly present with headache, nausea, vomiting, imbalance, and visual disturbances. Nerve compression may cause truncal ataxia, seizures and sensory deficits.

The imaging modality of choice is MRI with gadolinium contrast. Treatment usually consists of surgery, radiation, and chemotherapy.

MEDCOMIC®

THE MOST ENTERTAINING WAY TO STUDY MEDICINE

Third Edition



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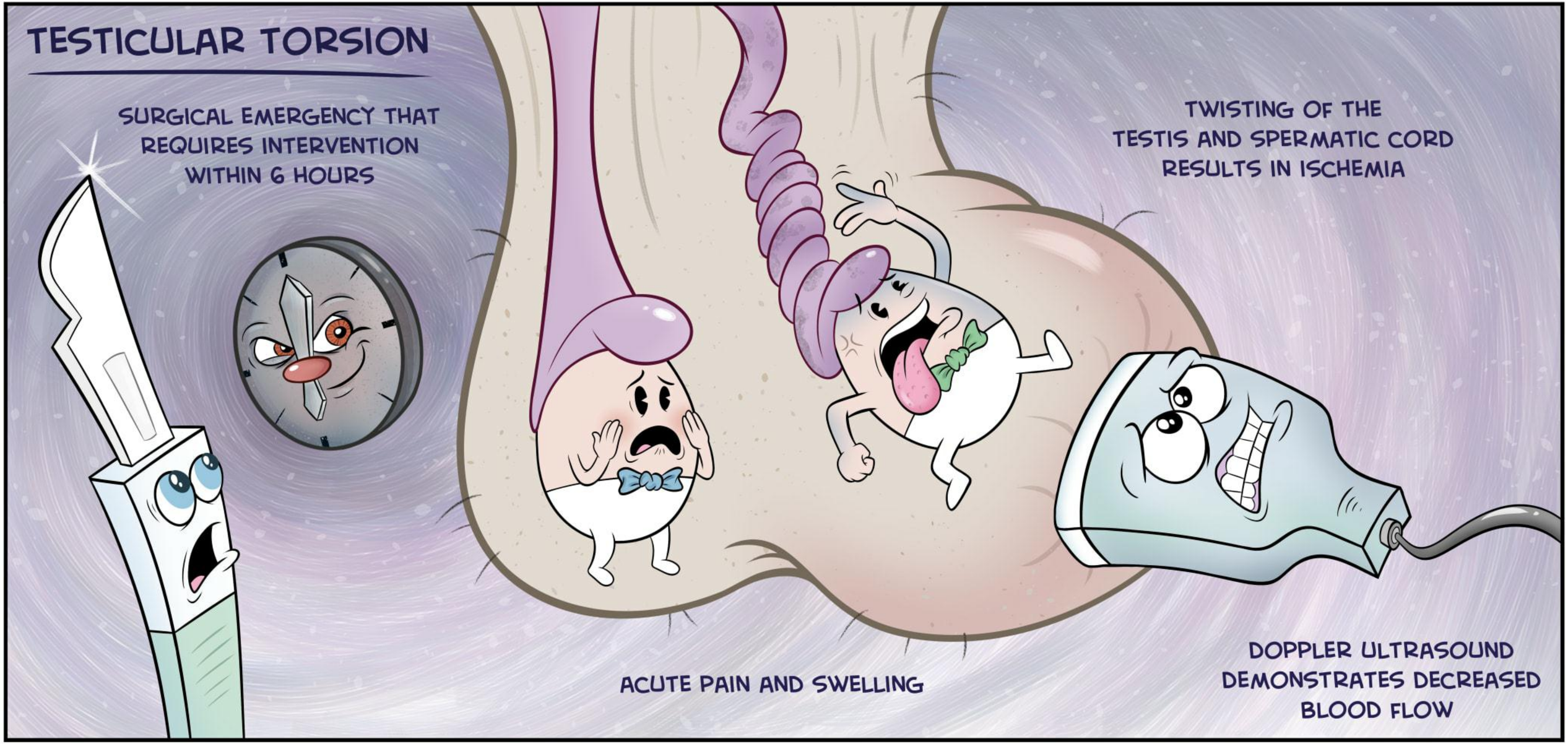
TESTICULAR TORSION
SURGICAL EMERGENCY THAT REQUIRES INTERVENTION WITHIN 6 HOURS
TWISTING OF THE TESTIS AND SPERMATIC CORD RESULTS IN ISCHEMIA
ACUTE PAIN AND SWELLING
DOPPLER ULTRASOUND DEMONSTRATES DECREASED BLOOD FLOW

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Testicular torsion occurs when the spermatic cord twists within the tunica vaginalis, resulting in ischemia to the epididymis and the testis. Testicular torsion may occur in the absence of a preceding event or after minor trauma. Patients typically experience acute severe pain that may be accompanied by nausea and vomiting.

On physical examination, the affected testis is edematous, tender to palpation, erythematous, and slightly elevated. The testis is also frequently found to be lying horizontally, displacing the epididymis so that it's not found in the normal posterolateral position. The unilateral absence of the cremasteric reflex is the most sensitive physical examination finding in testicular torsion.

Doppler ultrasound may be helpful in the diagnosis when there are equivocal clinical findings, but should not delay treatment. Immediate surgical intervention can salvage an ischemic testis if performed within six hours, but the success rate decreases significantly beyond 12 hours. Manual detorsion may also be performed in a similar manner to opening a book, in a medial to lateral motion.

AORTIC STENOSIS

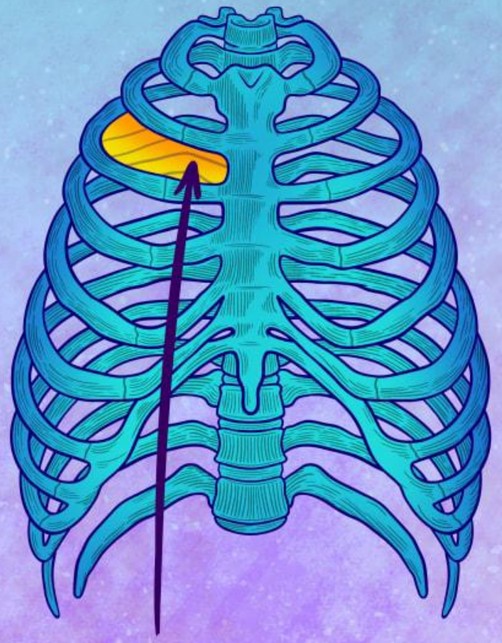
NARROWING AND CALCIFICATION OF THE AORTIC VALVE RESULTS IN LEFT VENTRICULAR OUTFLOW OBSTRUCTION

TESTS:

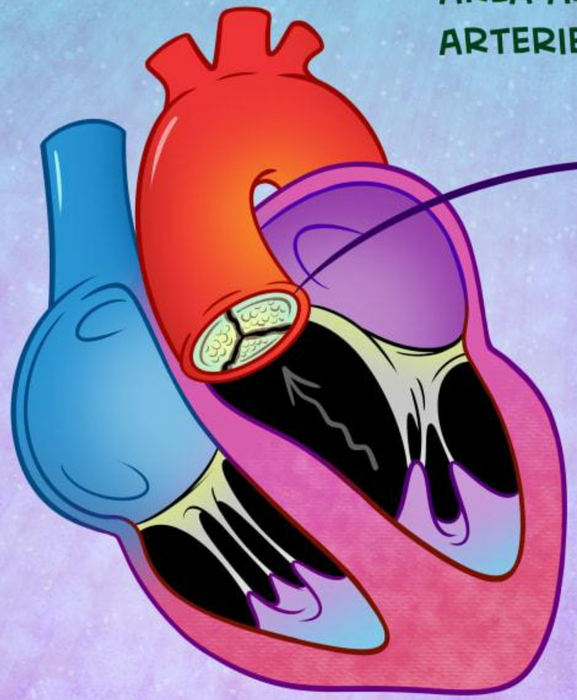
- ECG: LVH, ST-T WAVE CHANGES
- ECHO: CHECK STRUCTURES AND PRESSURE GRADIENT
- CARDIAC CATH: ASSESS VALVE AREA AND CORONARY ARTERIES

THREE MAIN CAUSES:

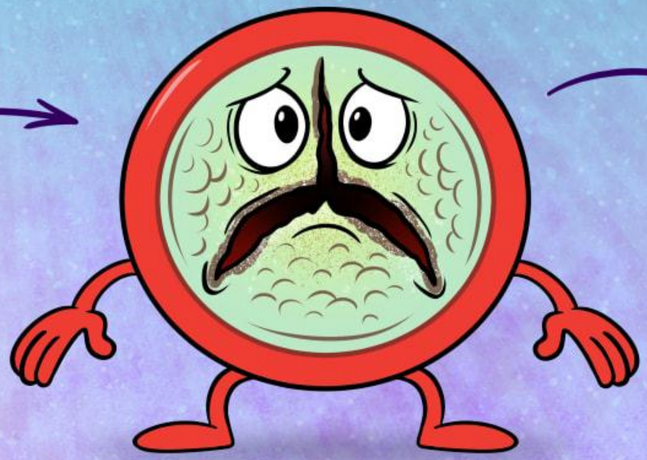
- CALCIFIC DISEASE OF A TRILEAFLET VALVE
- CALCIFICATION OF A BICUSPID VALVE
- RHEUMATIC VALVE DISEASE

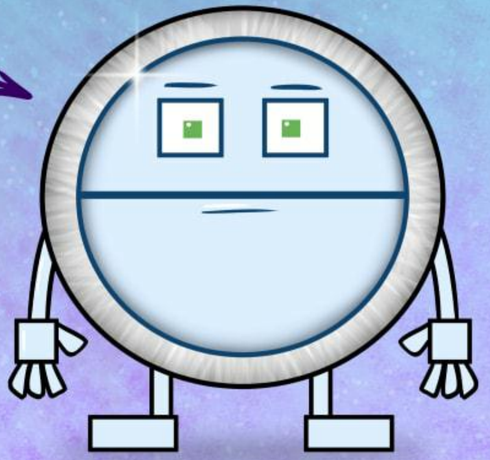


SYSTOLIC EJECTION MURMUR AT RIGHT SECOND INTERCOSTAL SPACE



POSSIBLE HEART FAILURE, ANGINA, AND SYNCOPE





INTERVENTION:

- SURGICAL AORTIC VALVE REPLACEMENT,
- TRANSCATHETER AORTIC VALVE REPLACEMENT
- BALLOON VALVULOPLASTY IN YOUNG PATIENTS OR POOR SURGICAL CANDIDATES

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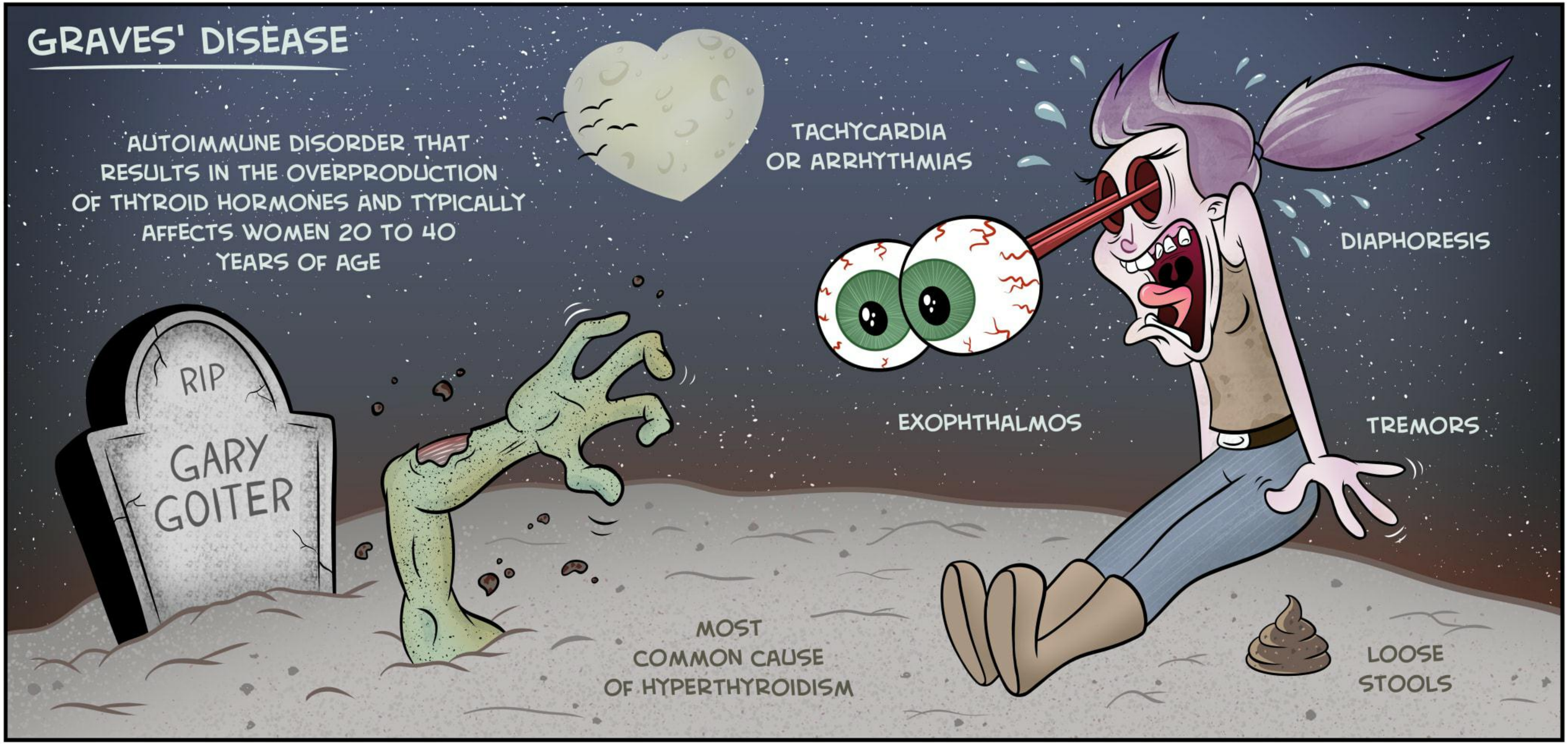
Causes of aortic stenosis (AS) include degenerative sclerosis with calcification of a trileaflet aortic valve, calcification of a congenital aortic bicuspid valve, or rheumatic fever. Narrowing of the aortic valve obstructs blood flow from the left ventricle to the ascending aorta during systole. Left untreated, patients with AS may develop heart failure, angina, syncope, exertional dyspnea, and arrhythmias.

A crescendo-decrescendo systolic murmur is heard at the right second intercostal space, which radiates to the carotids. Pulsus parvus et tardus is seen in severe AS, in which there is a weak and delayed carotid upstroke. Testing includes CXR, ECG, echocardiography, and cardiac catheterization to assess valve area and coronary arteries for atherosclerotic disease.

Asymptomatic adults with AS do not typically require intervention. Severe AS is treated with valve replacement or balloon valvuloplasty in young patients or poor surgical candidates.



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Graves' disease is an autoimmune disorder that results in the overproduction of thyroid hormones. It is the most common cause of hyperthyroidism. Women 20 to 40 years of age are typically affected. Thyrotoxicosis may cause unexplained weight loss due to an increased metabolic rate. Patients may present with goiter, exophthalmos, diaphoresis, tremors, heat intolerance, loose stools, and pretibial myxedema. The most common cardiac manifestation is tachycardia, often associated with palpitations. The treatment options for Graves' disease include antithyroid drugs, radioiodine therapy, and thyroidectomy.

WOUND CLOSURE

PRIMARY INTENTION: THE EDGES OF A CLEAN WOUND ARE CLOSED

SUTURES, STAPLES, STERI-STRIPS, OR GLUE MAY BE USED.

I'M WAITING FOR MY WOUNDS TO HEAL BY GRANULATION, EPITHELIALIZATION, AND CONTRACTION.

SECONDARY INTENTION: THE WOUND IS LEFT OPEN AND HEALS OVER TIME

IT IS MY INTENT TO PROPERLY CARE FOR THE WOUND BEFORE CLOSURE.

Dr. Acula

TERTIARY INTENTION (DELAYED PRIMARY CLOSURE): THE WOUND IS LEFT OPEN FOR A TIME AND THEN CLOSED

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The three types of wound healing:

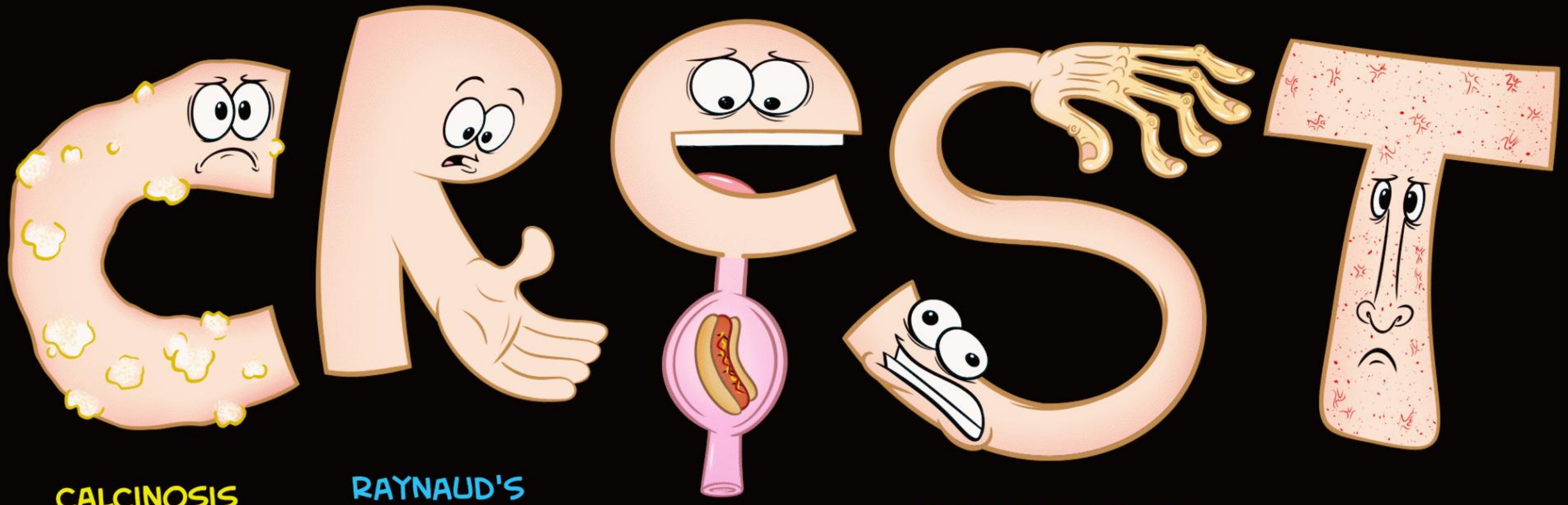
1. Primary intention – the wound closed immediately in some manner (e.g. suture, Steri-Strips, staples, glue)

2. Secondary intention – the wound is left open and allowed to heal by granulation, epithelialization, and contraction.

3. Tertiary intention (delayed primary closure) – the wound is left open and then later closed to allow for debridement and other types of wound care prior to closure.

CREST SYNDROME

LIMITED CUTANEOUS SYSTEMIC SCLEROSIS



CALCINOSIS CUTIS

RAYNAUD'S PHENOMENON

ESOPHAGEAL DYSMOTILITY

SCLERODACTYLY

TELANGIECTASIAS

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CREST syndrome (limited cutaneous systemic sclerosis):

Calcinosis: calcium deposits in the skin and tissues.

Raynaud's phenomenon: an exaggerated vascular response to cold temperature or emotional stress which may manifest as white-blue-red transitions in skin color.

Esophageal dysmotility: contractions in the esophagus become irregular or absent, causing dysphagia, heartburn, or atypical chest pain

Sclerodactyly: thickening and tightening of the skin, which makes it harder to bend or straighten the fingers. May also occur on the face.

Telangiectasias: dilated blood vessels which appear as red spots on the face and other areas.

RHABDOMYOLYSIS

MUSCLE TISSUE BREAKDOWN WITH RELEASE OF INTRACELLULAR CONTENTS (MYOGLOBIN) INTO CIRCULATION

ELEVATED CREATINE KINASE (CK) LEVELS

DARK, REDDISH BROWN URINE DUE TO MYOGLOBINURIA

MUSCLE PAIN AND WEAKNESS DUE TO TRAUMA

MYOGLOBIN MAY OCCLUDE THE STRUCTURES OF THE KIDNEY AND BREAK DOWN INTO TOXIC COMPOUNDS LEADING TO ACUTE TUBULAR NECROSIS OR ACUTE RENAL FAILURE

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Rhabdomyolysis is the breakdown of muscle tissue with release of intracellular contents such as myoglobin into the bloodstream. The condition usually follows major muscle trauma, especially a crush injury. It can also be caused by long-distance running, hyperthermia, infection, drugs, toxins, and electrolyte disturbances, among others.

Rhabdomyolysis is characterized by myalgias, weakness, and dark urine due to myoglobinuria. Muscle trauma may lead to compartment syndrome involving tissue hypoxia, muscle infarction, and neural damage. Creatine kinase (CK) levels are usually markedly elevated. Myoglobin may occlude the structures of the kidney and break down into toxic compounds leading to acute tubular necrosis or acute renal failure.

Management of rhabdomyolysis involves treating the underlying disorder, preventing renal failure by providing early and aggressive IV hydration, correcting electrolyte abnormalities, and recognizing and treating compartment syndrome if present.

COLLES' FRACTURE

DISTAL RADIUS FRACTURE WITH DORSAL ANGULATON AND IMPACTION

MAY BE SEEN IN YOUNG ATHLETES THAT SUSTAIN A HIGH ENERGY FALL

MOST COMMONLY SEEN IN ELDERLY WOMEN WITH OSTEOPOROTIC BONE THAT SUSTAIN A LOW ENERGY FALL

DINNER FORK DEFORMITY

FOOSH!

CRACK!

CAUSED BY A FALL ONTO A HYPEREXTENDED, RADIALLY DEVIATED WRIST WITH THE FOREARM IN PRONATION

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A colles' fracture is a fracture of the distal radius with dorsal angulation. The majority of these injuries demonstrate a "dinner fork" deformity. All displaced fractures should undergo closed reduction, which helps limit swelling, pain, and compression on the median nerve. Radiographic parameters for an acceptable reduction include less than 10 degrees of dorsal tilt, less than 2mm of articular step-off, and less than 5mm of radial shortening.

RENAL TUBULAR ACIDOSIS

AFFECTS THE RENAL TUBULES AND RESULTS IN A HYPERCHLOREMIC METABOLIC ACIDOSIS WITH A NORMAL SERUM ANION GAP

TYPE 2 PROXIMAL RTA

IMPAIRED BICARBONATE REABSORPTION

HIGH URINE pH INITIALLY, LATER < 5.5

HYPOKALEMIA

TYPE 1 DISTAL RTA

IMPAIRED HYDROGEN ION SECRETION

URINE pH > 5.5

HYPOKALEMIA

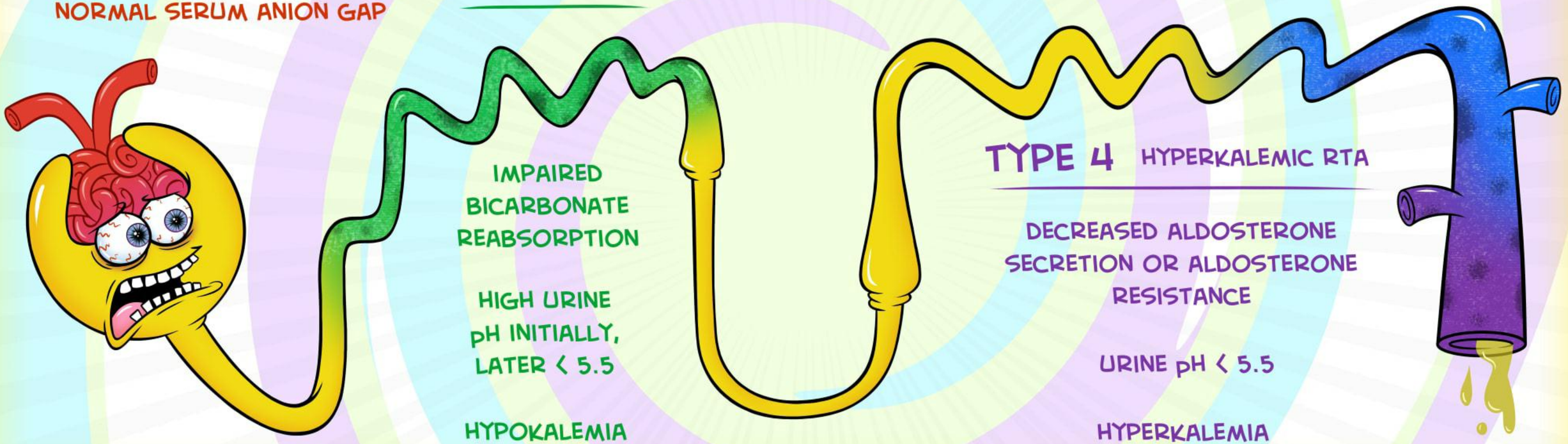
RENAL STONES

TYPE 4 HYPERKALEMIC RTA

DECREASED ALDOSTERONE SECRETION OR ALDOSTERONE RESISTANCE

URINE pH < 5.5

HYPERKALEMIA



BOWMAN'S CAPSULE

PROXIMAL TUBULE

LOOP OF HENLE

DISTAL TUBULE

COLLECTING DUCT

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Renal tubular acidosis (RTA) refers to a group of disorders affecting the renal tubules characterized by an impaired ability to acidify the urine and excrete acid. The condition results in a hyperchloremic metabolic acidosis with a normal serum anion gap.

Type 1 (distal) RTA is due to defective hydrogen ion secretion by alpha-intercalated cells in the late distal convoluted tubule and collecting duct. This leads to a buildup of hydrogen ions in the blood resulting in acidemia. Nephrolithiasis (calcium phosphate stones) is frequently associated with untreated type 1 RTA. Causes of type 1 RTA include Sjogren's syndrome, systemic lupus erythematosus, liver cirrhosis, and toxins (e.g. amphotericin B, lithium).

Type 2 (proximal) RTA is characterized by impaired bicarbonate reabsorption in the proximal convoluted tubule. This leads to an increase in bicarbonate loss in the urine and increased acidity of the blood. Type 2 RTA is associated with multiple myeloma, Fanconi syndrome, and toxins (e.g. Acetazolamide, outdated tetracycline).

A combined pathology of proximal and distal tubule dysfunction leading to type 3 RTA is very rare and the causes are poorly understood.

Type 4 (hyperkalemic) RTA is due to aldosterone deficiency or aldosterone resistance in the collecting ducts. Causes include Addison's disease, diabetic nephropathy, sickle cell disease, and drugs (e.g. trimethoprim, NSAIDs, ACE inhibitors, spironolactone).

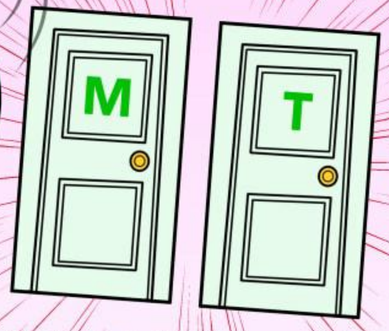
HEART SOUNDS

OCCUR DUE TO VIBRATIONS PRODUCED BY VALVULAR ACTIONS AND BLOOD FLOW WITHIN THE HEART

S4.

ATRIAL CONTRACTION AND DECREASED VENTRICULAR COMPLIANCE DURING LATE DIASTOLE

S1!

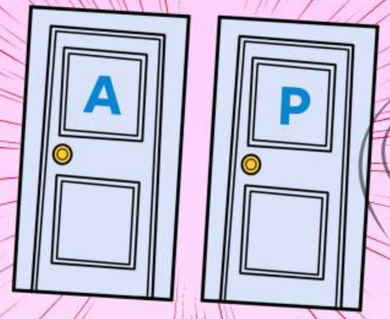


LUB!

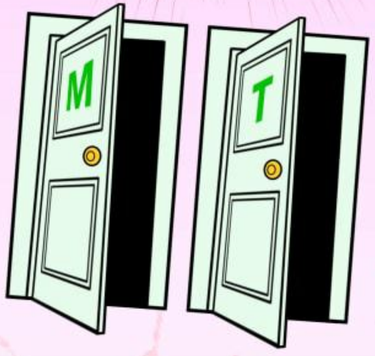
MITRAL AND TRICUSPID VALVE CLOSURE

SYSTOLE

AORTIC AND PULMONIC VALVE CLOSURE



DUB!



S2!

S3.

RAPID VENTRICULAR FILLING DURING EARLY DIASTOLE

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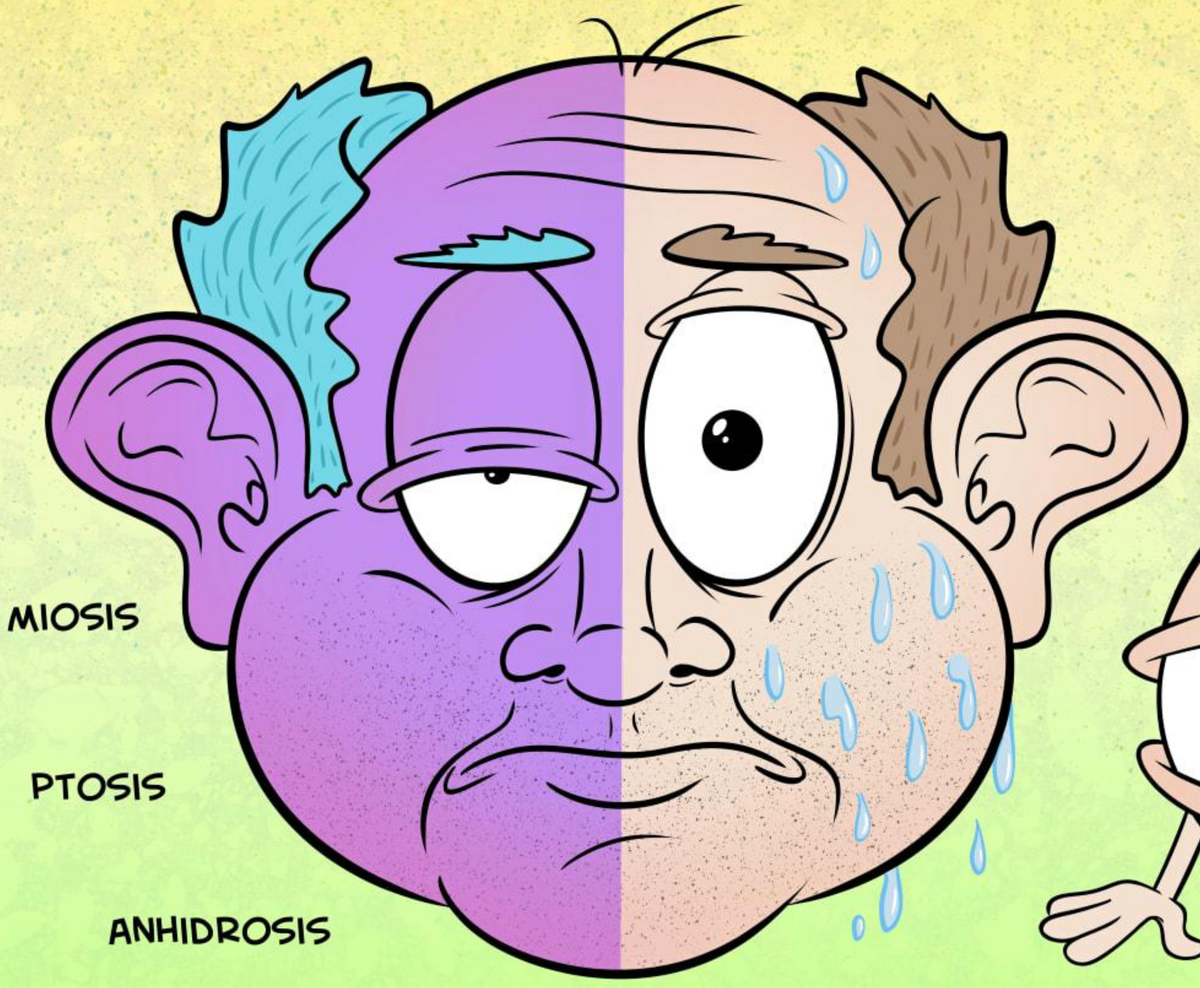
The first heart sound (S1) represents mitral and tricuspid valve closure as the ventricular pressure exceeds atrial pressure at the beginning of systole.

The second heart sound (S2) represents aortic and pulmonary valve closure at the end of systole/beginning of diastole. The second heart sound is physiologically split because the aortic valve normally closes before the pulmonic valve. It is normal for the split of S2 to widen during inspiration and narrow during expiration.

The third heart sound (S3) represents rapid ventricular filling during early diastole. The third heart sound can be a normal finding in children and young athletes. In older adults S3 is associated with heart failure.

The fourth heart sound (S4) occurs during late diastole, just before S1. It represents the vibration of a stiff ventricle as it's forced to receive blood during atrial contraction.

HORNER SYNDROME



MIOSIS

PTOSIS

ANHIDROSIS

SO APPARENTLY WE'RE ABOUT TO GET COCAINE POURED ALL OVER US.

I'M GOOD WITH COKE.

THE DIAGNOSIS CAN ALSO BE MADE WITH APRACLONIDINE...

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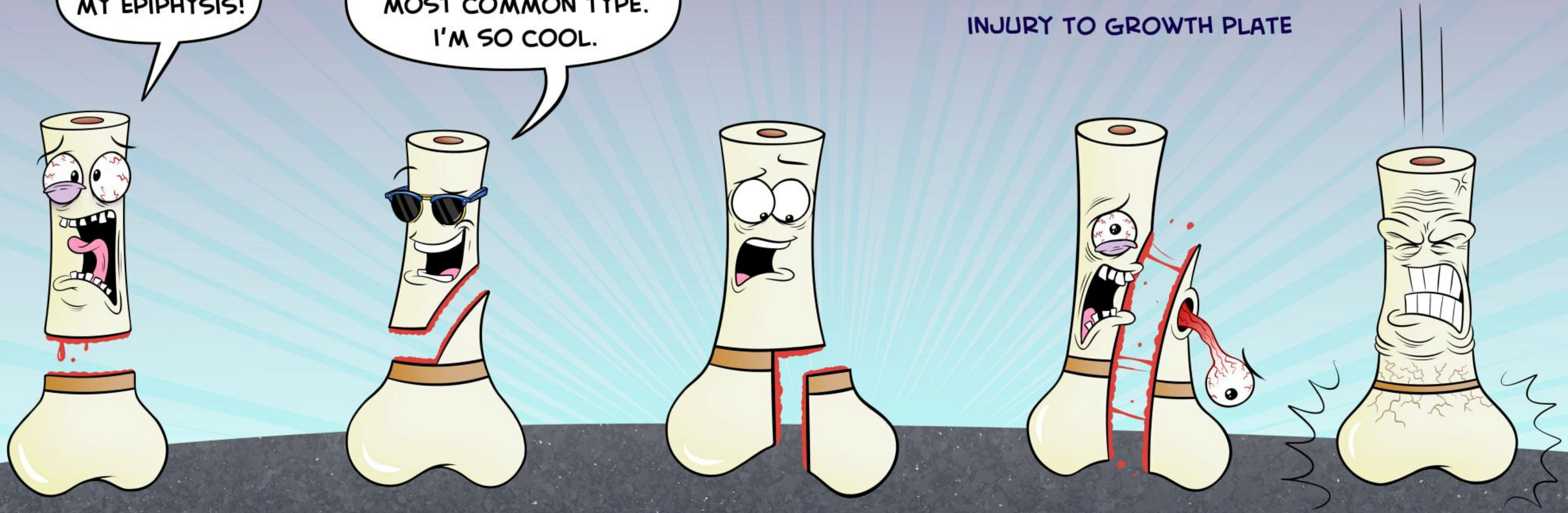
Horner syndrome results from disruption of the sympathetic pathway that runs from the hypothalamus to the eye. Classic signs include miosis, ptosis, and anhidrosis of the affected side.

Causes include brain tumor, brain stem infarct, pancoast tumor, cervical adenopathy, skull and neck trauma, carotid dissection, and thoracic aortic aneurysm.

Instilling cocaine or apraclonidine drops into both eyes can confirm the diagnosis of Horner syndrome. Imaging studies such as MRI, CT, or MRA may be obtained based on the history and physical exam. Treatment depends on the underlying cause of Horner syndrome.

SALTER-HARRIS FRACTURES

INJURY TO GROWTH PLATE



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Pediatric physeal fractures have traditionally been described by the five-part Salter-Harris classification system.

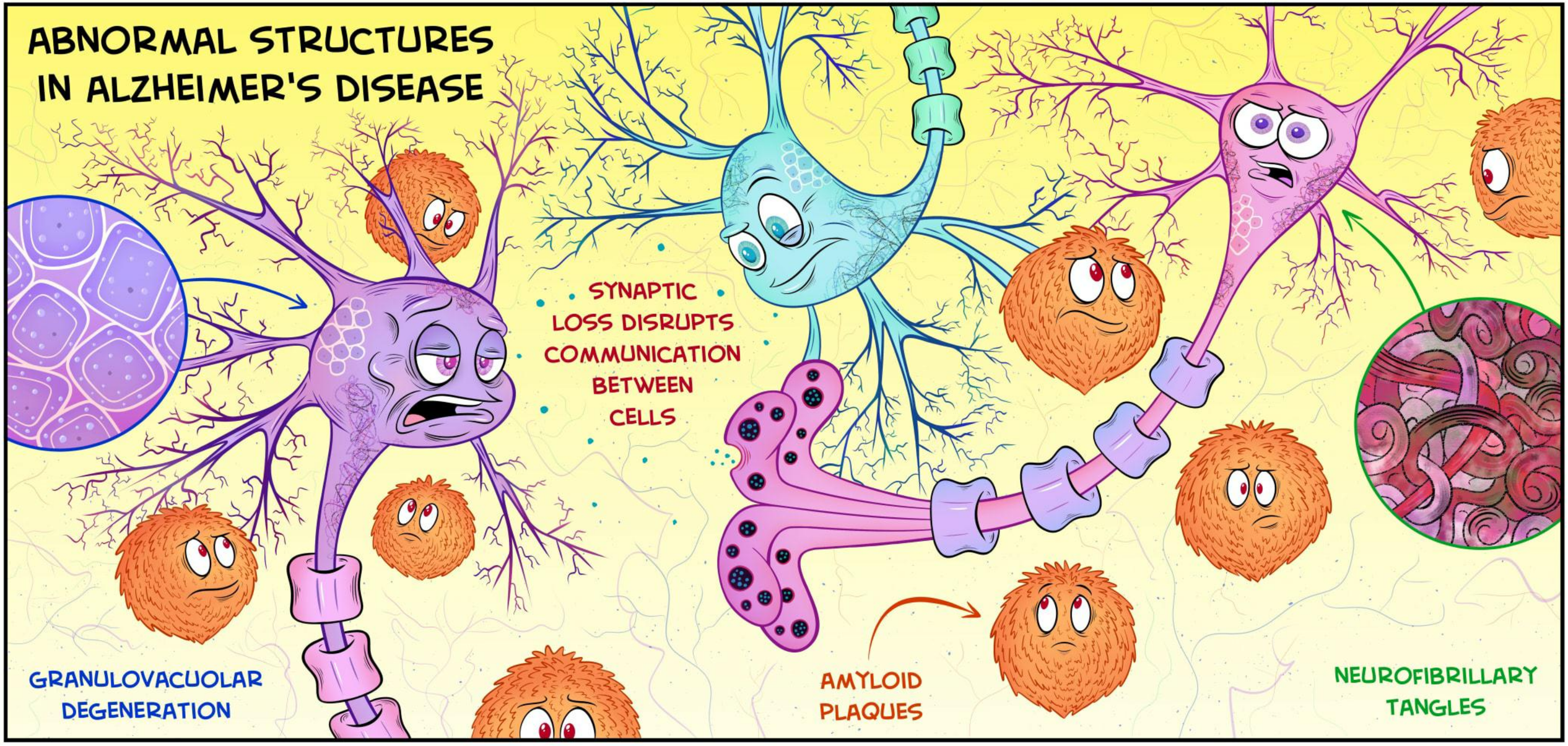
Type I fractures occur through the growth plate. These injuries may present with normal radiographs and the diagnosis is often made clinically when tenderness is palpated over the growth plate.

Type II fractures occurs through the growth plate and metaphysis. Type II injuries are the most common physeal fractures.

Type III fractures occur through the growth plate and epiphysis. Type III injuries are intra-articular and the diagnosis is made radiographically based on the appearance of an epiphyseal fragment not associated with a metaphyseal fracture.

Type IV fractures are also intra-articular and occur through the growth plate, metaphysis and epiphysis.

Type V fractures occur when there is a crush injury of the growth plate. The prognosis is poor because growth arrest and partial physeal closure are common.



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Alzheimer's disease (AD) is a neurodegenerative disorder and the most common cause of dementia. Neurons communicate via chemical messages passed between two cells across a small gap called a *synapse*. In AD, abnormal cellular structures lead to a loss of synapses and neurons, which results in atrophy of the brain.

The pathogenesis is largely unknown, but there are several characteristic abnormalities found in the brain tissue of patients with AD. These features include excessive granulovacuoles, amyloid plaques, and neurofibrillary tangles.

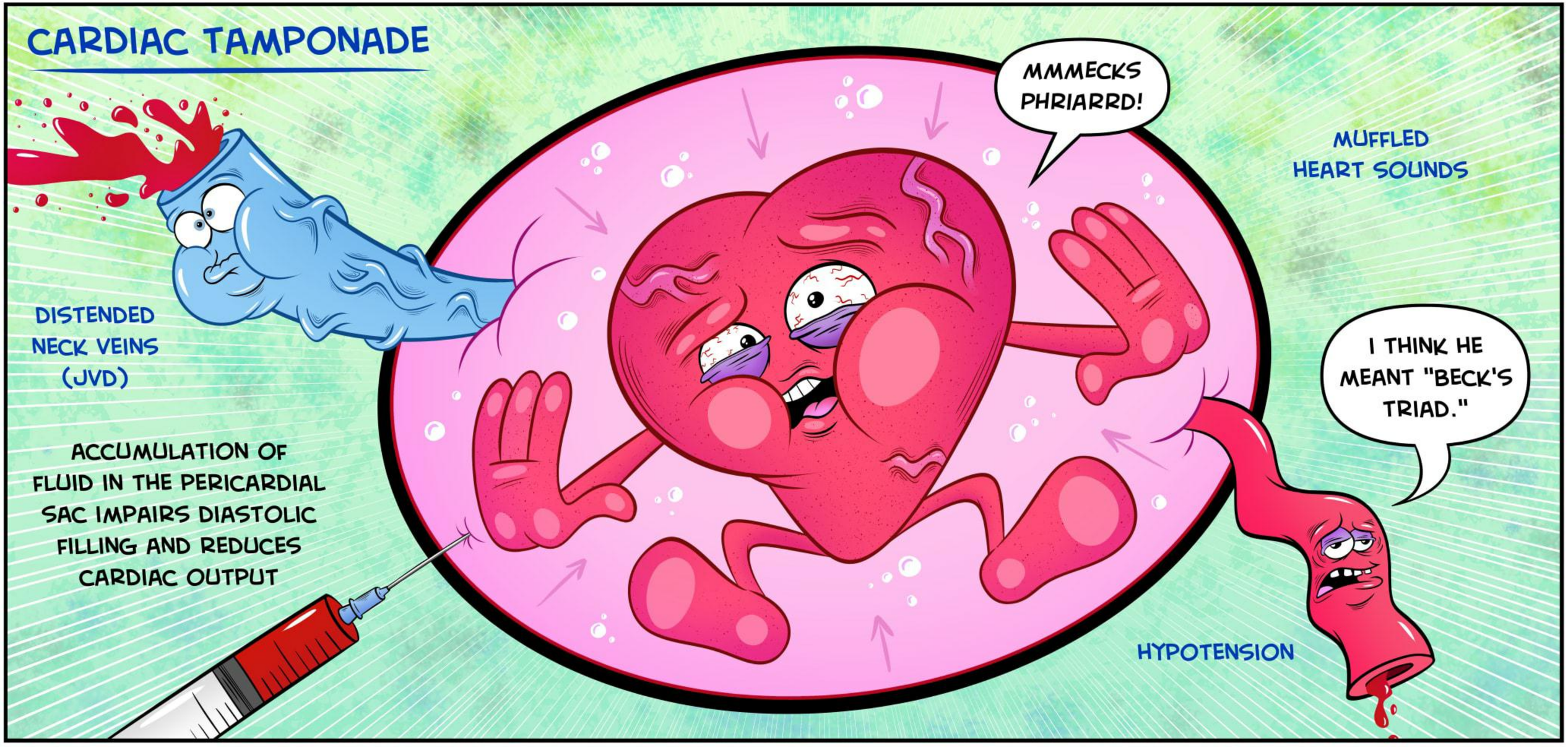
The nucleus basalis of Meynert (nbM) is a basal forebrain structure that houses the brain's largest collection of cholinergic neurons. In AD the nbM suffers severe neuron loss and decreased production of acetylcholine, a neurotransmitter with an important role in learning and memory.

Granulovacuolar degeneration is found within the cytoplasm of neurons of the hippocampus. An abnormally high number of fluid-filled spaces, called vacuoles, enlarge the cell's body causing neuronal dysfunction or cell death.

Amyloid plaques form outside of neurons when protein pieces called beta-amyloid clump together. The plaques block cell-to-cell signaling at synapses and may trigger processes that lead to nerve cell death.

Neurofibrillary tangles are abnormal collections of protein threads inside the nerve that twist around each other. The main protein composing neurofibrillary tangles is called *tau*.

CARDIAC TAMPONADE


MMMECKS PHRIARRD!
I THINK HE MEANT "BECK'S TRIAD."
DISTENDED NECK VEINS (JVD)
ACCUMULATION OF FLUID IN THE PERICARDIAL SAC IMPAIRS DIASTOLIC FILLING AND REDUCES CARDIAC OUTPUT
MUFFLED HEART SOUNDS
HYPOTENSION

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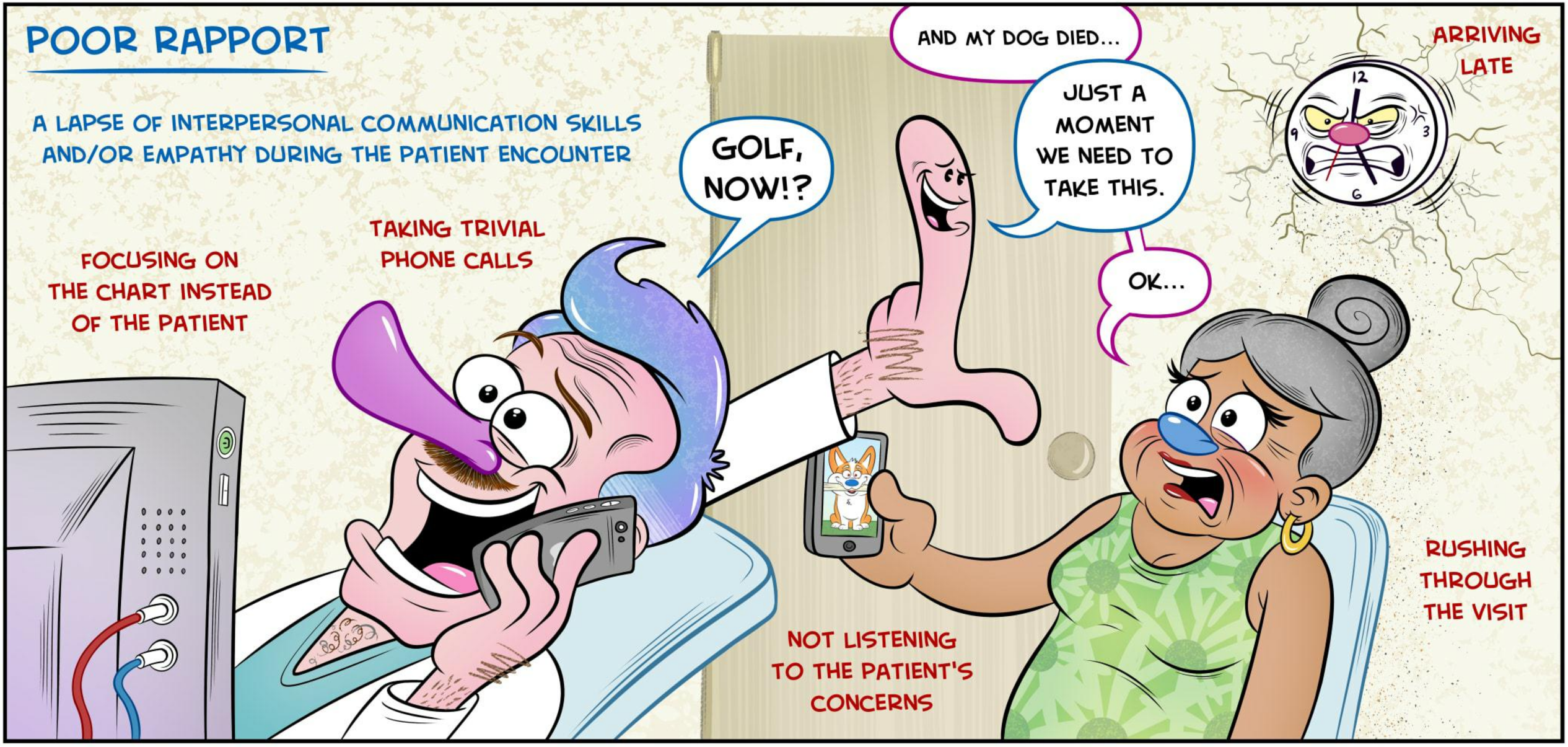
Cardiac tamponade is an accumulation of fluid in the pericardial sac that compresses the heart, impairs diastolic filling, and leads to a reduction in cardiac output.

Tamponade is most often caused by penetrating trauma. Other causes of acute cardiac tamponade include aortic rupture and procedures such as pacer placement. Subacute cardiac tamponade can occur in the setting of malignancy or pericarditis.

Beck's triad is a collection of three signs associated with cardiac tamponade: distended neck veins, muffled heart sounds, and hypotension. Pulsus paradoxus is also suggestive, consisting of a decrease in systolic BP of > 10 mmHg during inspiration.

Patients suspected of having cardiac tamponade should be evaluated with EKG, chest radiograph, and echocardiography. EKG may demonstrate low voltage and sinus tachycardia. Electrical alternans consists of beat-to-beat shifts in the QRS complex due to swinging of the heart in a large effusion.

Treatment consists of the removal of pericardial fluid to relieve the elevated intrapericardial pressure and improve cardiac output. This can be achieved with pericardiocentesis (needle aspiration of the pericardial cavity), pericardial window (surgical creation of an opening), or pericardectomy (resection of a portion or all of the pericardium).


POOR RAPPORT
A LAPSE OF INTERPERSONAL COMMUNICATION SKILLS AND/OR EMPATHY DURING THE PATIENT ENCOUNTER
FOCUSING ON THE CHART INSTEAD OF THE PATIENT
TAKING TRIVIAL PHONE CALLS
GOLF, NOW!?
AND MY DOG DIED...
JUST A MOMENT WE NEED TO TAKE THIS.
OK...
ARRIVING LATE
NOT LISTENING TO THE PATIENT'S CONCERNS
RUSHING THROUGH THE VISIT

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SCOLIOSIS

LATERAL CURVATURE OF THE SPINE

A COBB ANGLE OF 10 DEGREES OR MORE DEFINES SCOLIOSIS

MODERATE CURVES (20 TO 40 DEGREES) ARE TREATED WITH PT AND BRACING

COBB ANGLE

SEVERE CURVES GREATER THAN 40 DEGREES MAY REQUIRE SURGERY

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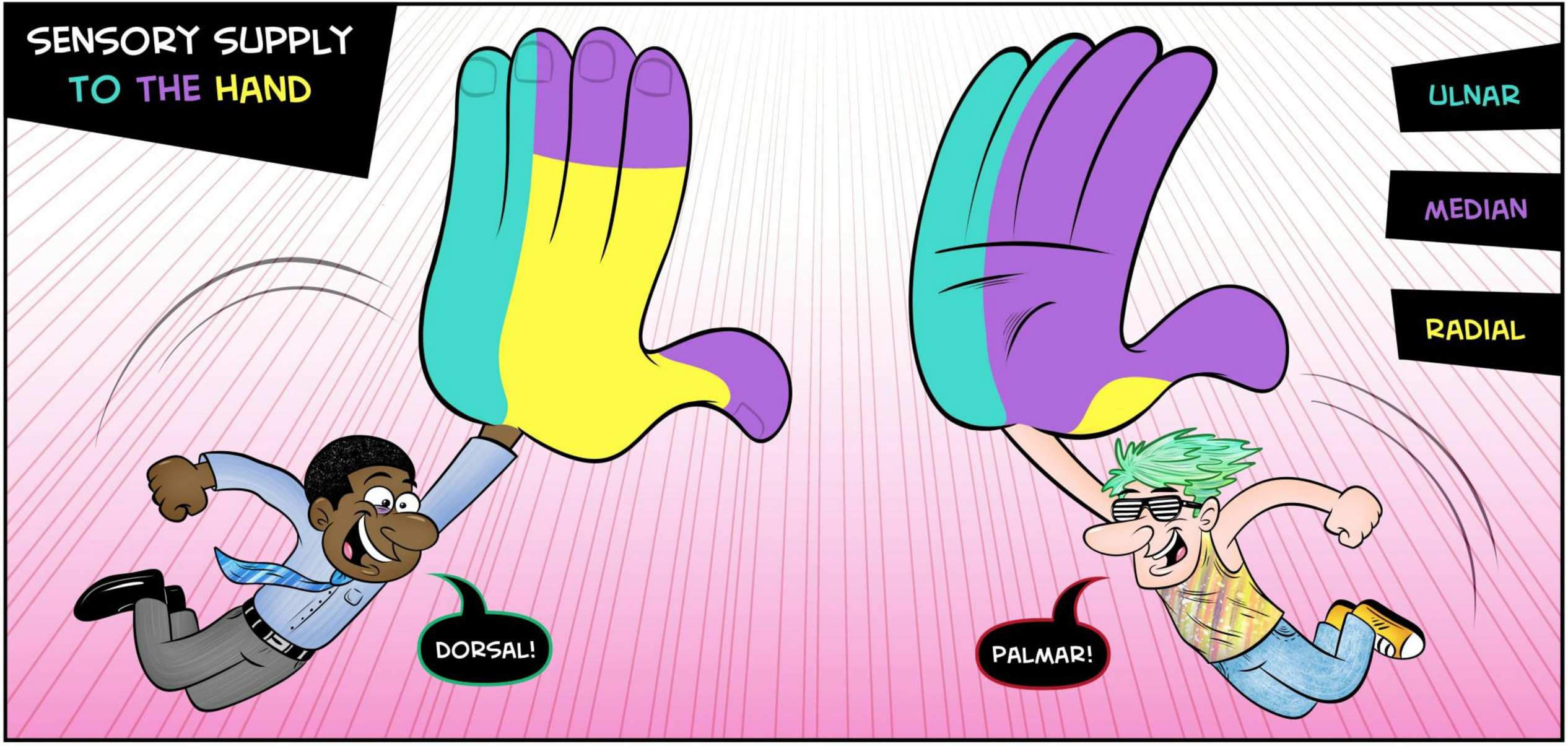

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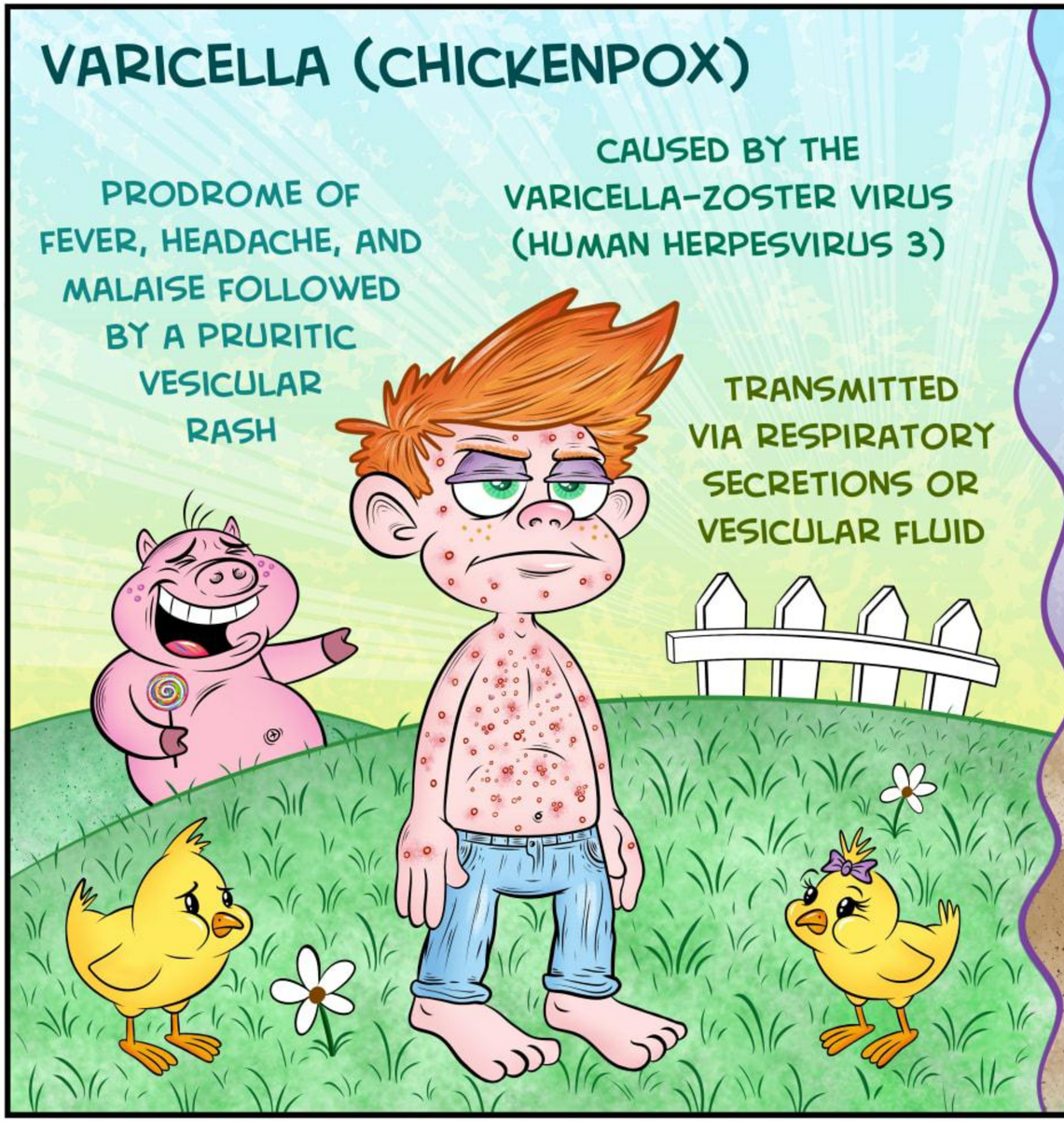
VARICELLA (CHICKENPOX)

PRODROME OF FEVER, HEADACHE, AND MALAISE FOLLOWED BY A PRURITIC VESICULAR RASH

CAUSED BY THE VARICELLA-ZOSTER VIRUS (HUMAN HERPESVIRUS 3)

TRANSMITTED VIA RESPIRATORY SECRETIONS OR VESICULAR FLUID

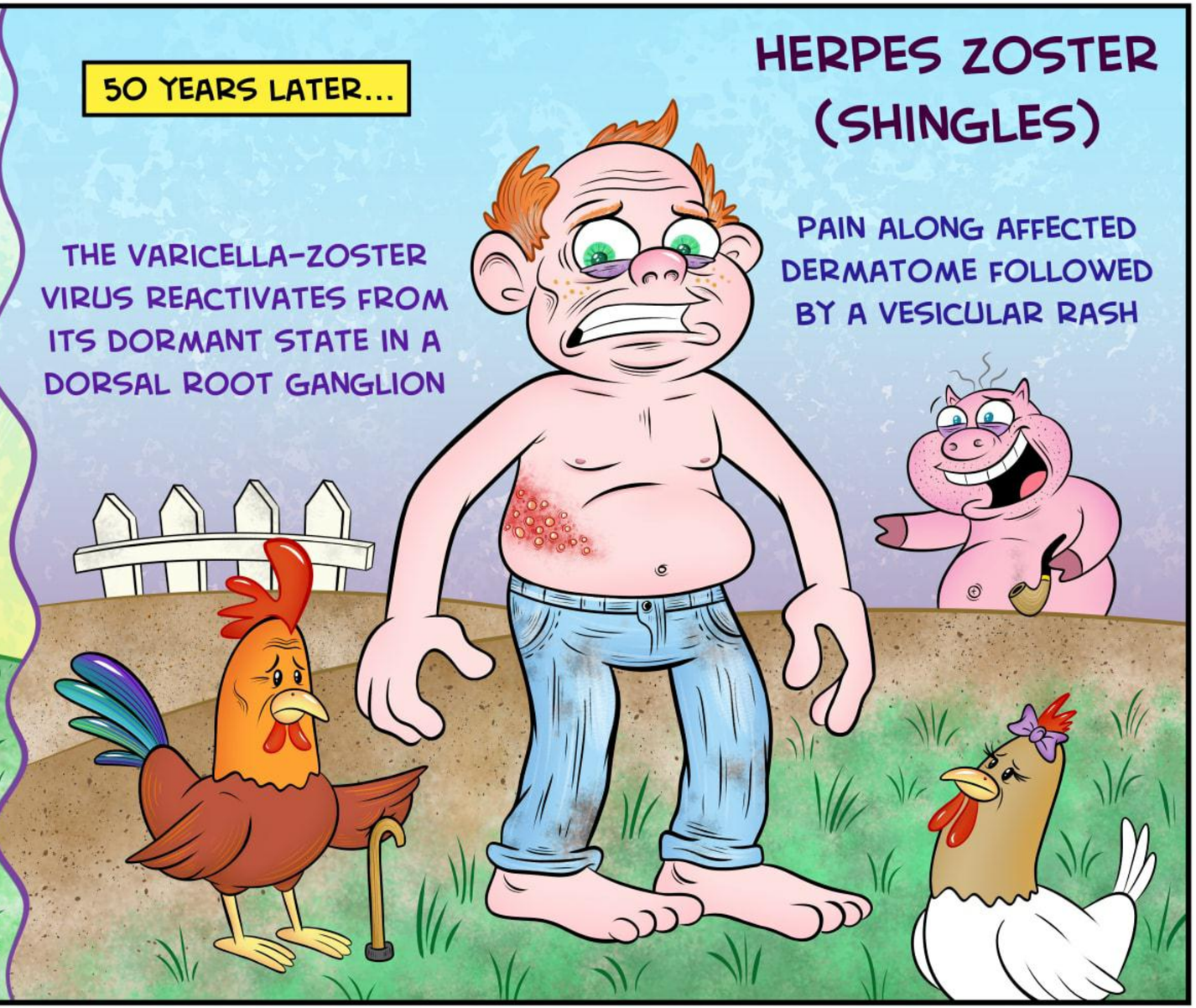


50 YEARS LATER...

HERPES ZOSTER (SHINGLES)

THE VARICELLA-ZOSTER VIRUS REACTIVATES FROM ITS DORMANT STATE IN A DORSAL ROOT GANGLION

PAIN ALONG AFFECTED DERMATOME FOLLOWED BY A VESICULAR RASH



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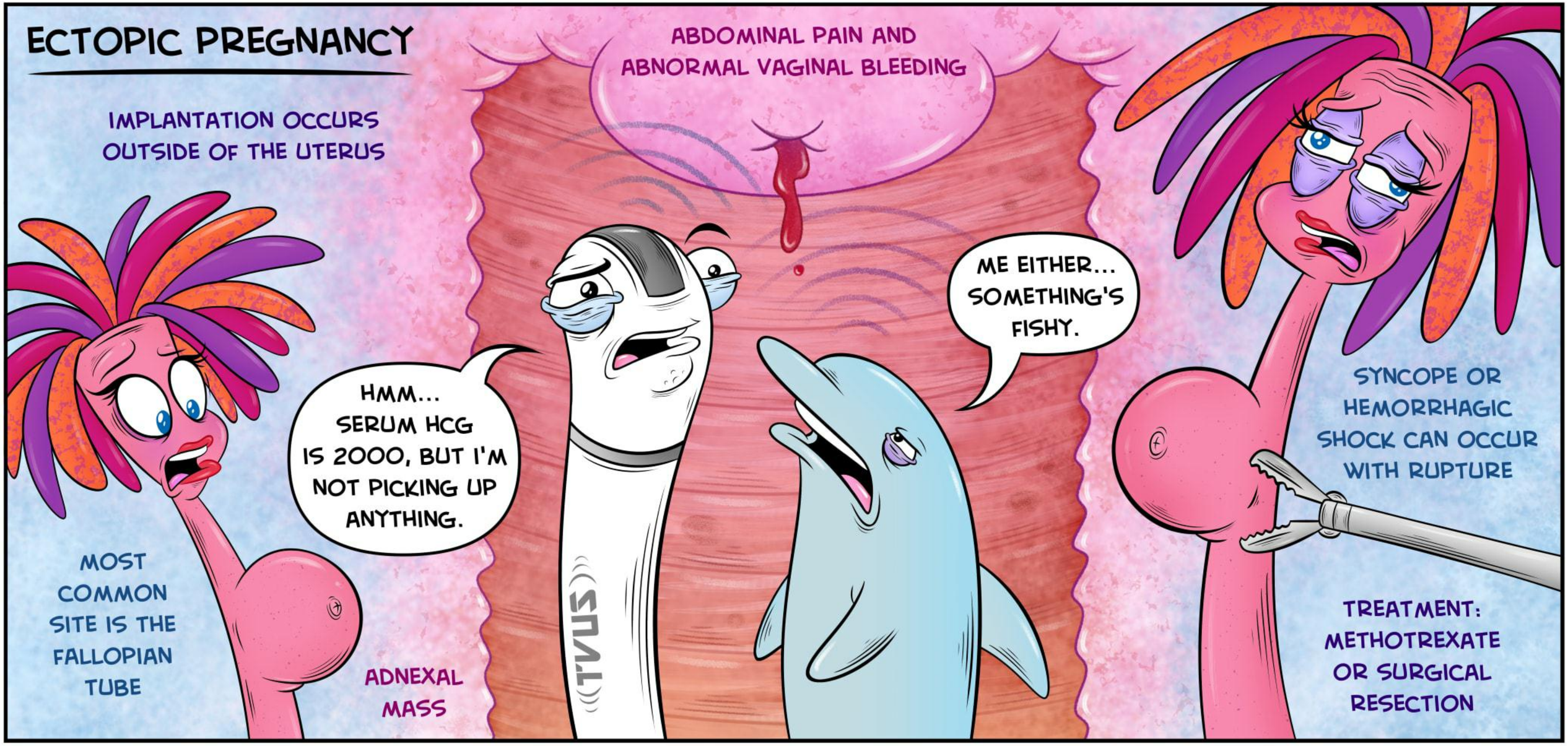
ECTOPIC PREGNANCY
IMPLANTATION OCCURS OUTSIDE OF THE UTERUS
MOST COMMON SITE IS THE FALLOPIAN TUBE
HMM... SERUM HCG IS 2000, BUT I'M NOT PICKING UP ANYTHING.
ADNEXAL MASS
ABDOMINAL PAIN AND ABNORMAL VAGINAL BLEEDING
TVUS
ME EITHER... SOMETHING'S FISHY.
SYNCOPE OR HEMORRHAGIC SHOCK CAN OCCUR WITH RUPTURE
TREATMENT: METHOTREXATE OR SURGICAL RESECTION

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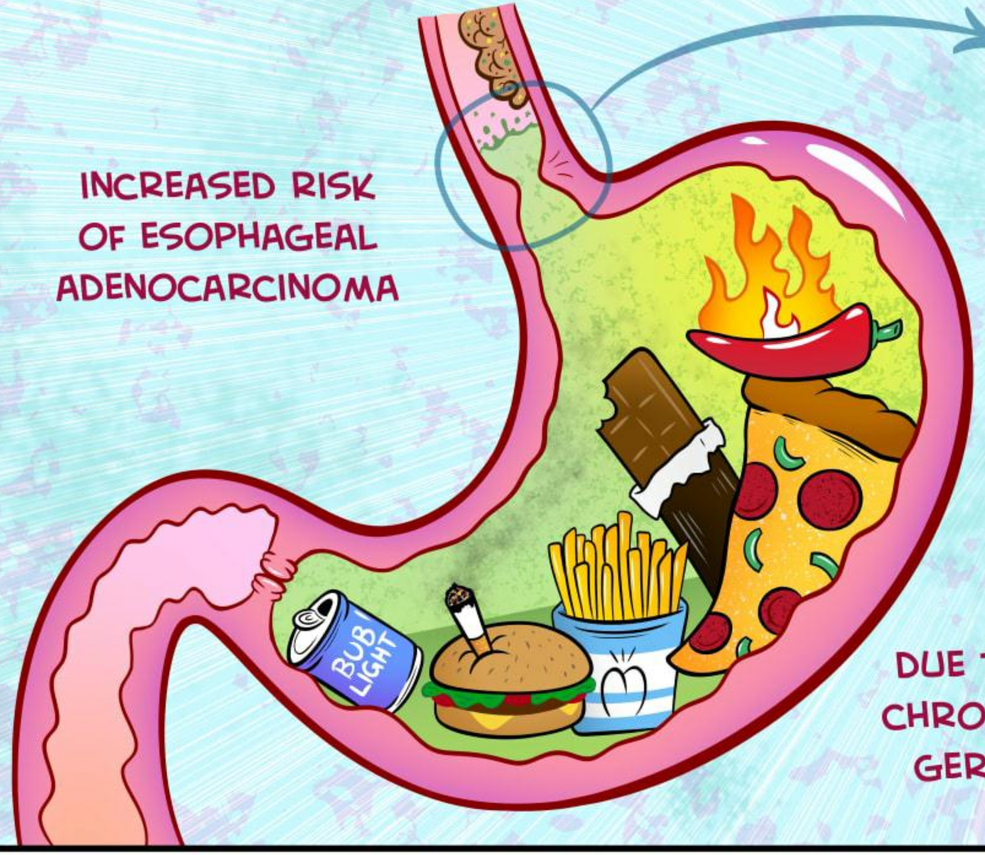
BARRETT ESOPHAGUS

METAPLASTIC COLUMNAR EPITHELIUM REPLACES THE STRATIFIED SQUAMOUS EPITHELIUM THAT NORMALLY LINES THE DISTAL ESOPHAGUS

INCREASED RISK OF ESOPHAGEAL ADENOCARCINOMA



DUE TO CHRONIC GERD

MANAGED WITH ENDOSCOPIC SURVEILLANCE

LES DYSFUNCTION

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NORMAL PRESSURE HYDROCEPHALUS

GRADUAL ACCUMULATION OF CSF CAUSES ENLARGED BRAIN VENTRICLES

?

DEMENTIA

MAGNETIC GAIT

URINARY INCONTINENCE

NORMAL OR SLIGHTLY ELEVATED CSF PRESSURE

TREATMENT WITH VENTRICULOPERITONEAL SHUNTING MAY IMPROVE SYMPTOMS

BRAIN IMAGING SHOWS VENTRICULAR ENLARGEMENT WITHOUT CORTICAL ATROPHY

LUMBAR PUNCTURE WITH REMOVAL OF 30-50 ML OF CSF HELPS CONFIRM THE DIAGNOSIS

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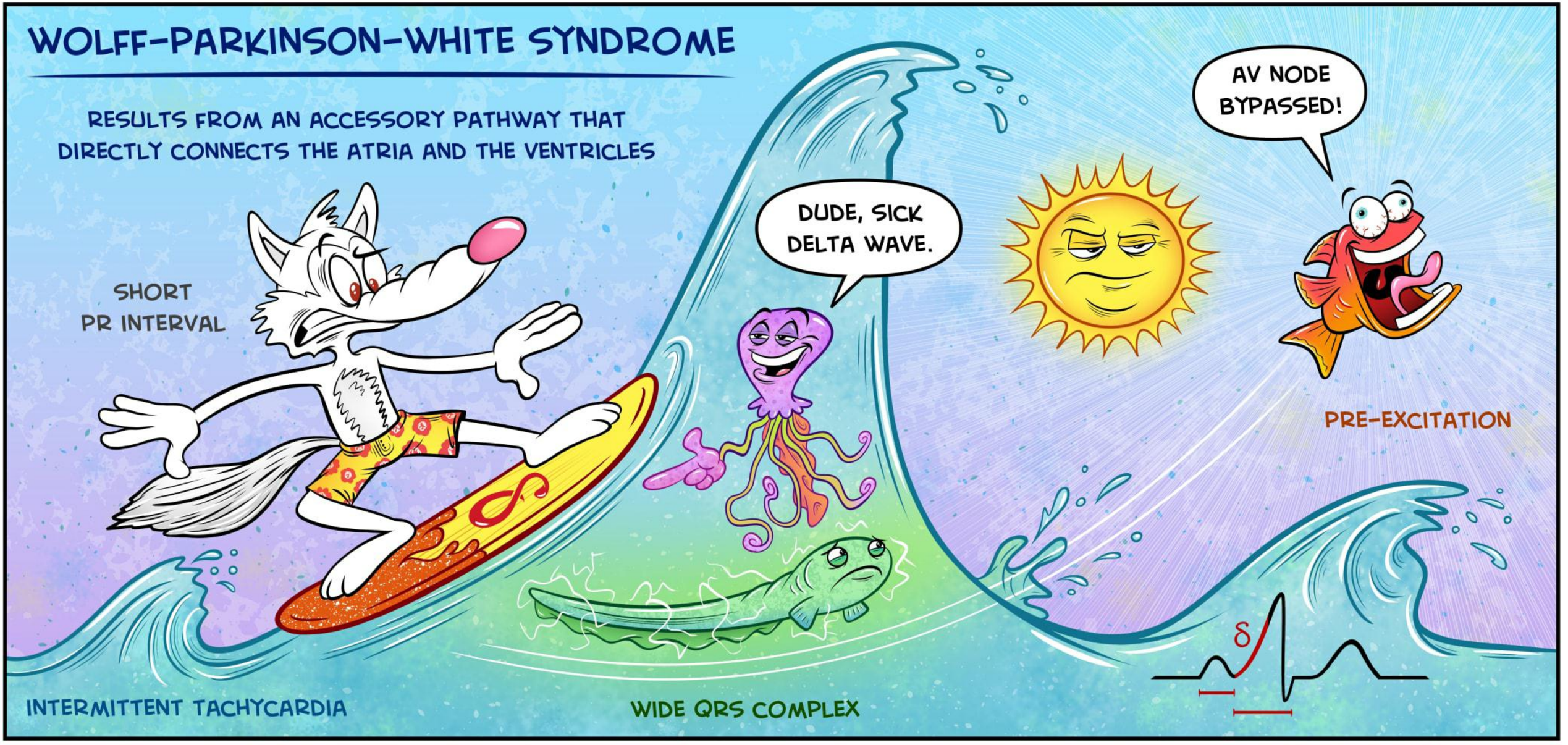
WOLFF-PARKINSON-WHITE SYNDROME

RESULTS FROM AN ACCESSORY PATHWAY THAT DIRECTLY CONNECTS THE ATRIA AND THE VENTRICLES



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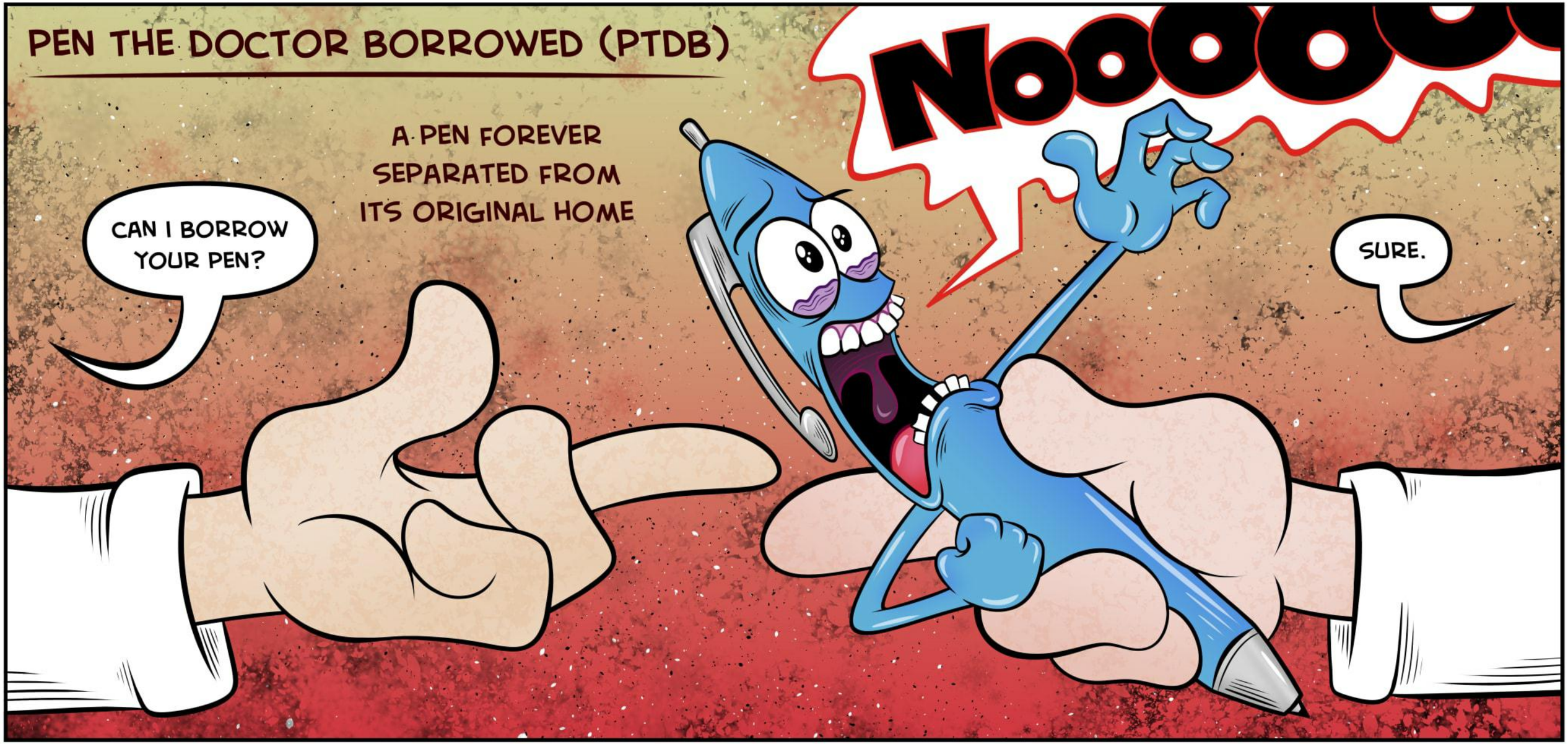

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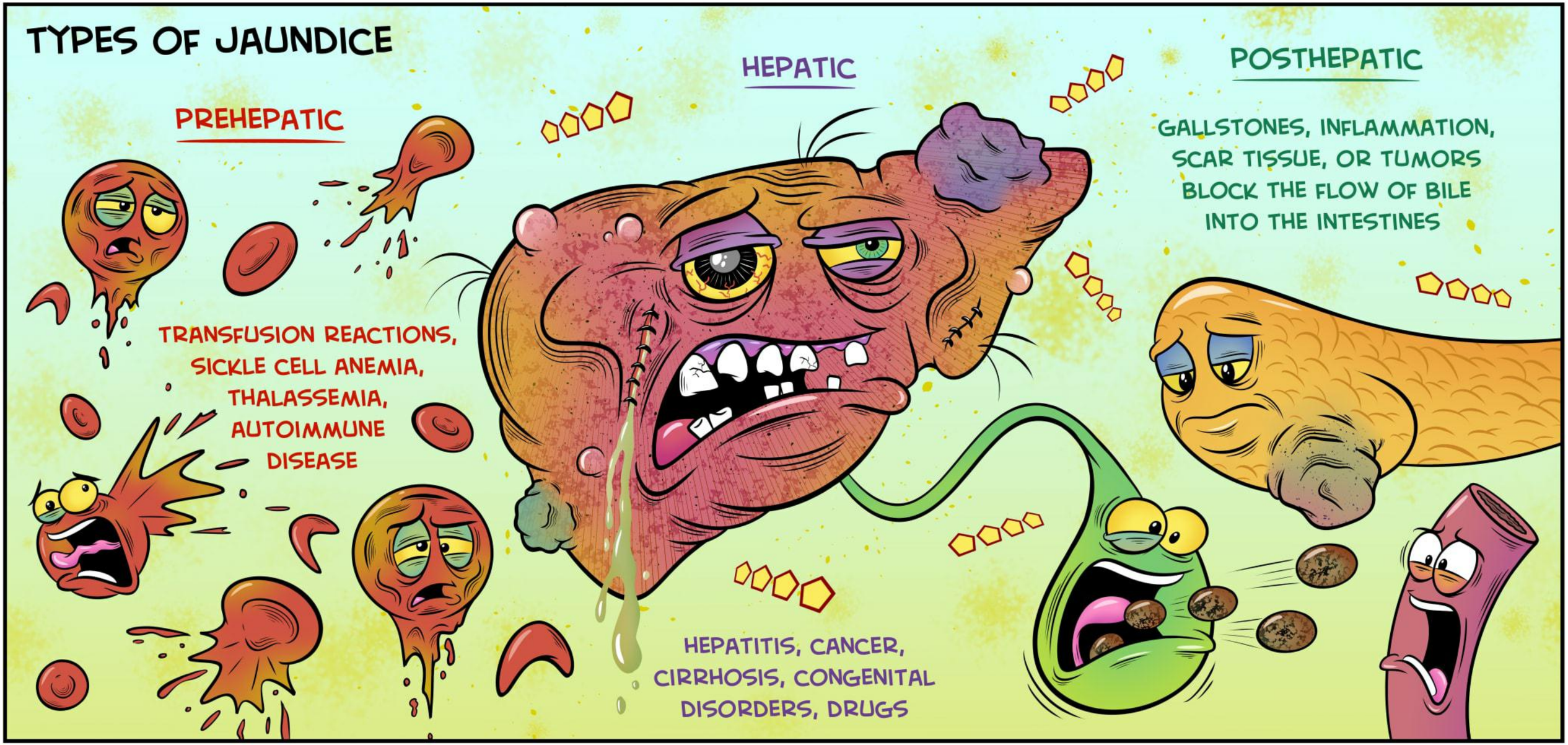
TYPES OF JAUNDICE
PREHEPATIC
TRANSFUSION REACTIONS, SICKLE CELL ANEMIA, THALASSEMIA, AUTOIMMUNE DISEASE
HEPATIC
HEPATITIS, CANCER, CIRRHOSIS, CONGENITAL DISORDERS, DRUGS
POSTHEPATIC
GALLSTONES, INFLAMMATION, SCAR TISSUE, OR TUMORS BLOCK THE FLOW OF BILE INTO THE INTESTINES

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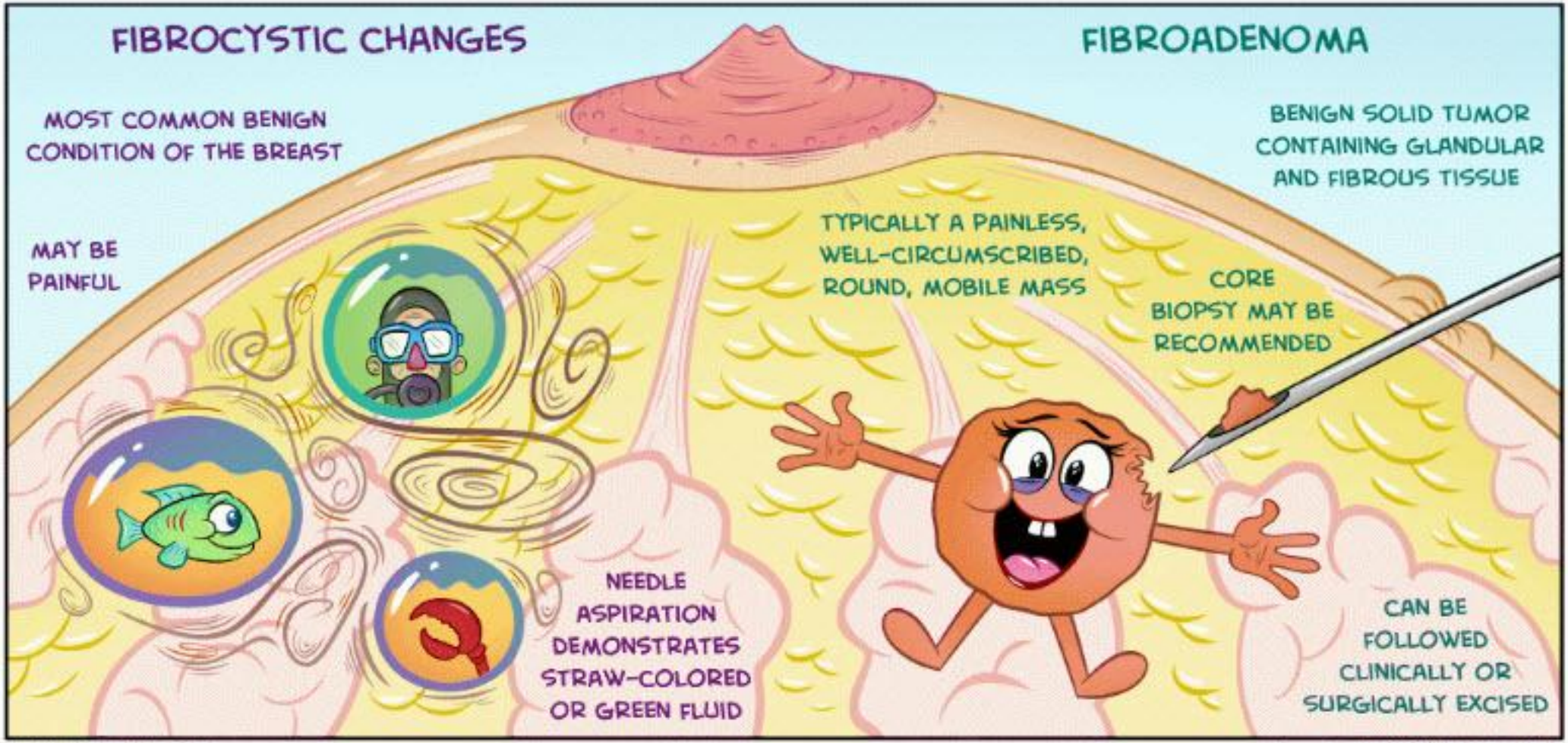
FIBROCYSTIC CHANGES
MOST COMMON BENIGN CONDITION OF THE BREAST
MAY BE PAINFUL
NEEDLE ASPIRATION DEMONSTRATES STRAW-COLORED OR GREEN FLUID
FIBROADENOMA
BENIGN SOLID TUMOR CONTAINING GLANDULAR AND FIBROUS TISSUE
TYPICALLY A PAINLESS, WELL-CIRCUMSCRIBED, ROUND, MOBILE MASS
CORE BIOPSY MAY BE RECOMMENDED
CAN BE FOLLOWED CLINICALLY OR SURGICALLY EXCISED

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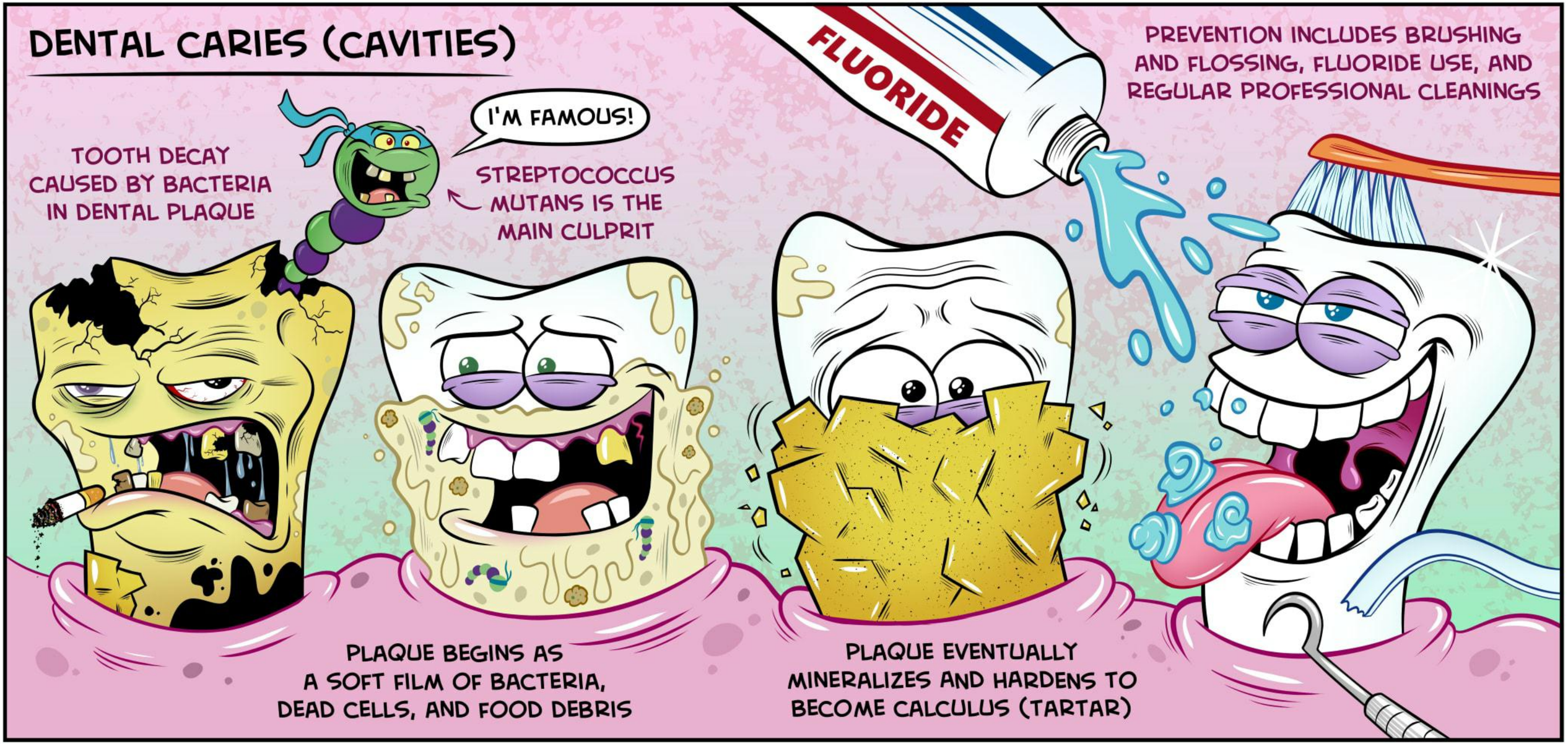
DENTAL CARIES (CAVITIES)


TOOTH DECAY CAUSED BY BACTERIA IN DENTAL PLAQUE
I'M FAMOUS!
STREPTOCOCCUS MUTANS IS THE MAIN CULPRIT
PLAQUE BEGINS AS A SOFT FILM OF BACTERIA, DEAD CELLS, AND FOOD DEBRIS
PLAQUE EVENTUALLY MINERALIZES AND HARDENS TO BECOME CALCULUS (TARTAR)
FLUORIDE
PREVENTION INCLUDES BRUSHING AND FLOSSING, FLUORIDE USE, AND REGULAR PROFESSIONAL CLEANINGS

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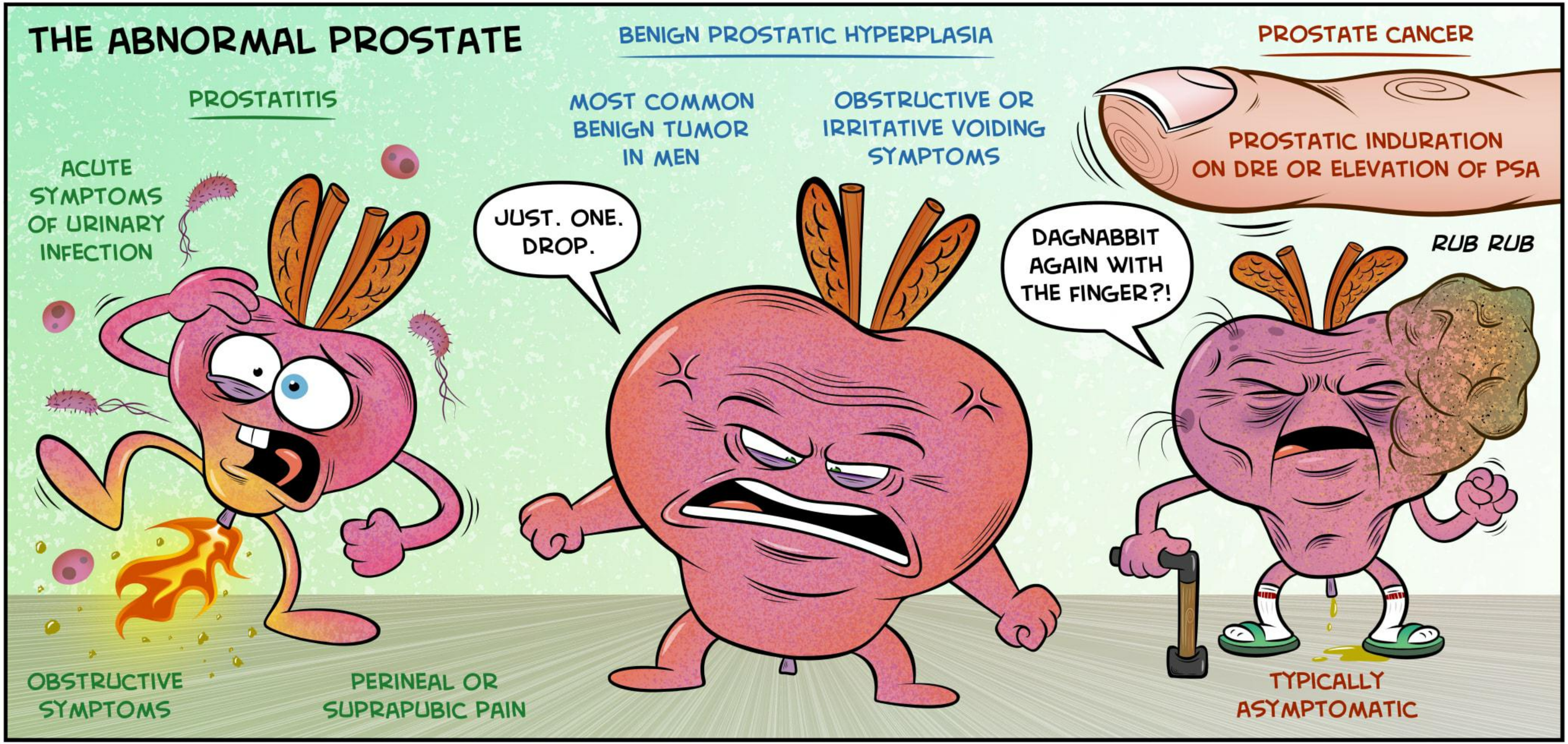

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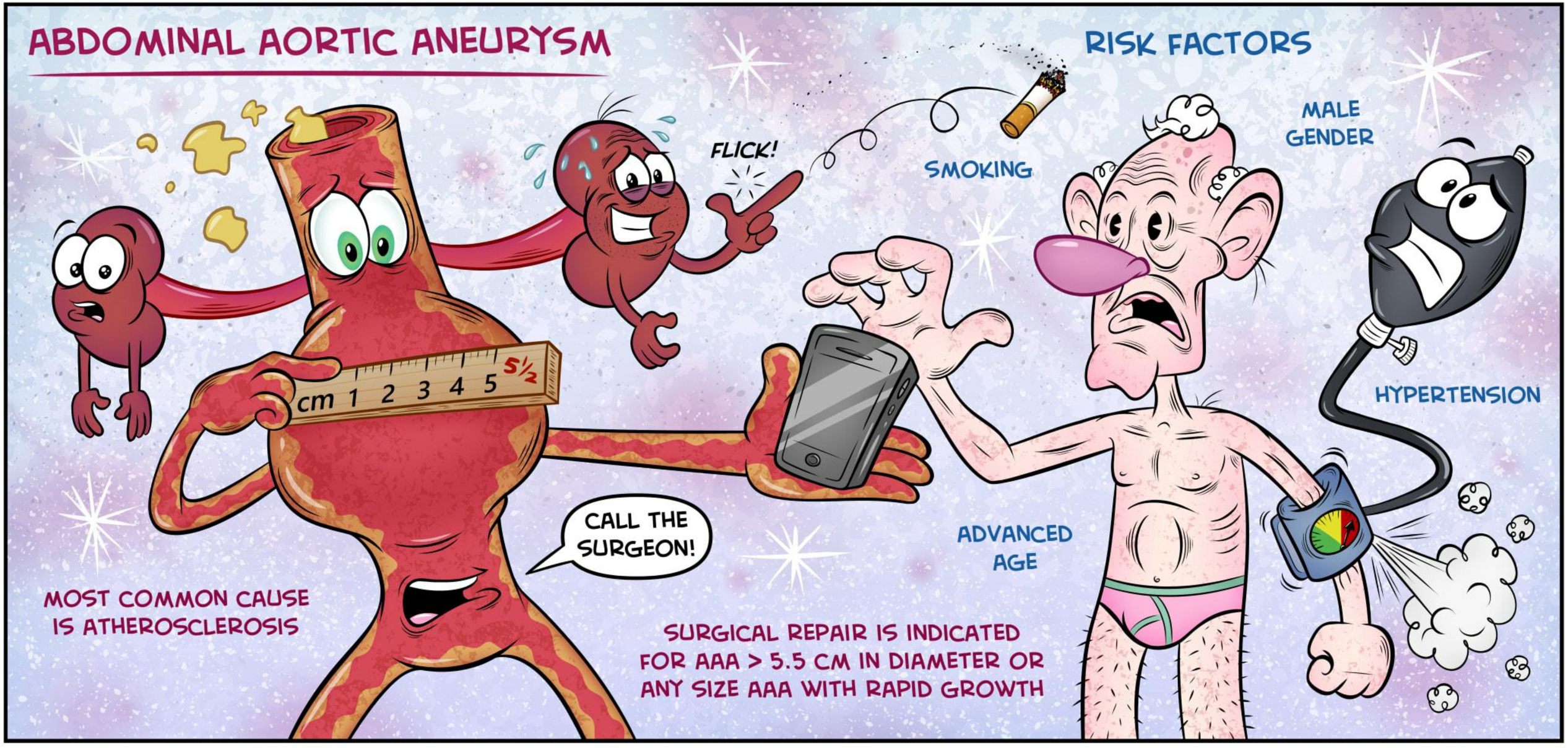
ABDOMINAL AORTIC ANEURYSM


RISK FACTORS
FLICK!
SMOKING
MALE GENDER
HYPERTENSION
cm 1 2 3 4 5 5½
CALL THE SURGEON!
ADVANCED AGE
MOST COMMON CAUSE IS ATHEROSCLEROSIS
SURGICAL REPAIR IS INDICATED FOR AAA > 5.5 CM IN DIAMETER OR ANY SIZE AAA WITH RAPID GROWTH

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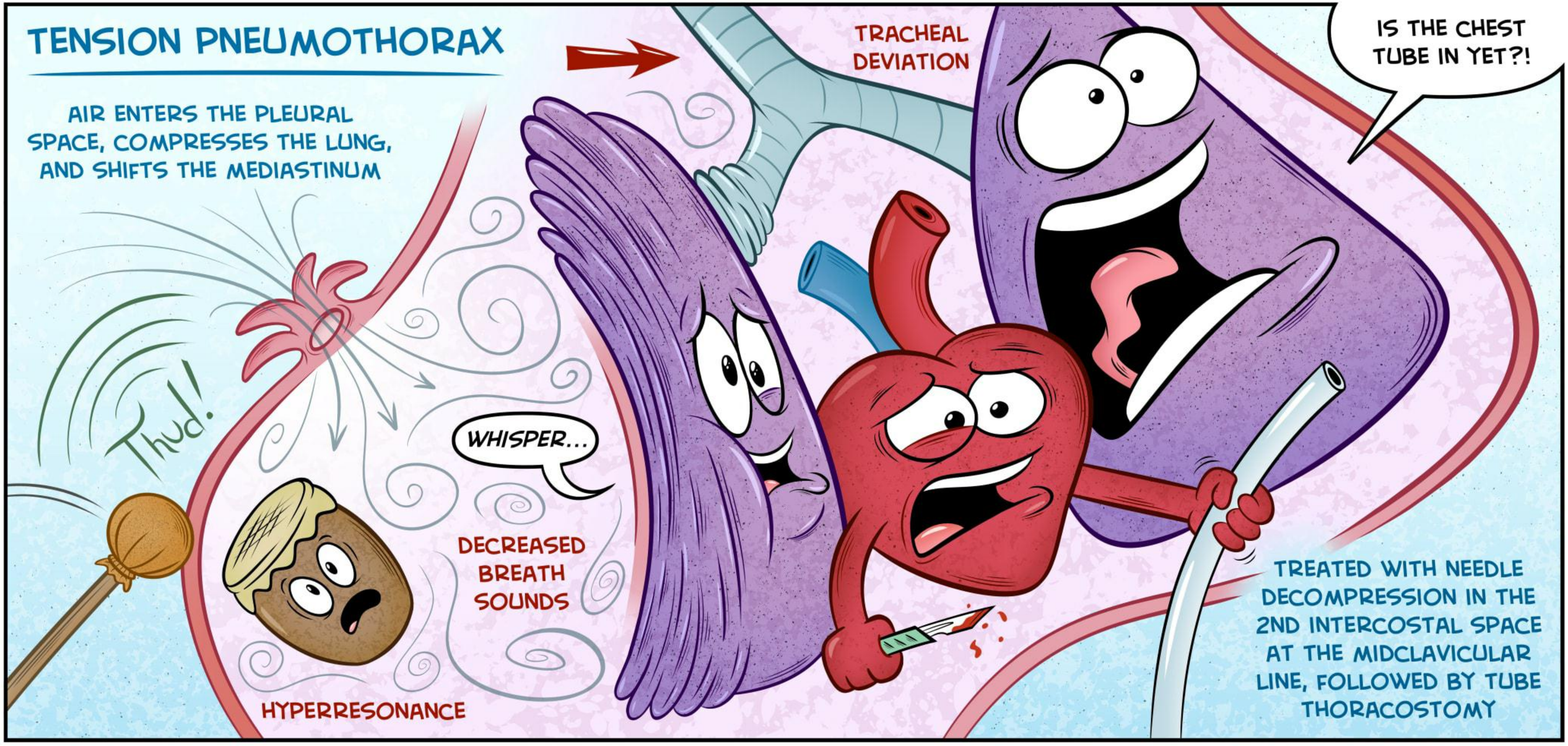
TENSION PNEUMOTHORAX
AIR ENTERS THE PLEURAL SPACE, COMPRESSES THE LUNG, AND SHIFTS THE MEDIASTINUM
Thud!
WHISPER...
DECREASED BREATH SOUNDS
HYPERRESONANCE
TRACHEAL DEVIATION
IS THE CHEST TUBE IN YET?!
TREATED WITH NEEDLE DECOMPRESSION IN THE 2ND INTERCOSTAL SPACE AT THE MIDCLAVICULAR LINE, FOLLOWED BY TUBE THORACOSTOMY

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